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REVIEW OF THE AVAILABLE LITERATURE OF THE PHARYNX AND PHARYNGEAL SURGERY FOR 1955.*

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ANATOMY.

Batson[1], as usual, presented a most excellent anatomic description of the cricopharyngeus muscle, together with consideration of its functions. Constriction of the cricopharyngeus muscle is well recognized by all endoscopists, and the sphincteric occlusion just below the pyriform sinuses adds to the problem of esophagoscopy. This constriction merits the name "cricopharyngeal pinchcock," given to it by Jackson. Closure is tonic and constant; opening is coordinate and momentary, for deglutition and emesis. The illustrations presented from cadaveric material are unexcelled. This article is difficult to summarize, and should be read in its entirety to be fully appreciated.

Brunner[2] was intrigued with the fact that there is no definite anatomic division between the hypopharynx and cervical esophagus. A detailed study of the regional anatomy, together with roentgenologic visualization, revealed interesting information. It is well known that a triangular area of weakness exists between the oblique fibers of the inferior constrictor muscle and the transverse fibers of the cricopharyn-

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geus muscle. Brunner stated that in dissection of the cricopharyngeus muscle the oblique and transverse fibers are indistinguishable. It is contended that this sphincter muscle of the hypopharynx undergoes active contraction and forms the "hypopharyngeal bar," which is analogous to Passavant's bar in the epipharynx. This hypopharyngeal bar serves as the borderline between the hypopharynx and cervical esophagus, and is located at the level of the inferior border of the cricoid cartilage. Brunner believed that a well-developed hypopharyngeal bar could conceivably be responsible occasionally for the sensation of a lump in the throat.

In a study of the pharyngeal orifice of the Eustachian tube in the aged, Keleman[3] utilized cadaveric material from individuals ranging in age from 61 to 79 years. He summarized his study by stating that the anterior and posterior folds of the pharyngeal orifice were found to represent almost solid glandular blocks with a minimum of lymphatic tissue in one-half of the bases in the anterior and no lymphatic tissue at all in the posterior fold. In both poor vascularization prevailed. This disappearance of lymphatic tissue with advancing age from this area forms a contrast to conditions existing around the palatine tonsils.

Barton's[4] discussion of the significance of the ridge of Passavant should be of prime interest to all otolaryngologists, particularly those who perform many adenotonsillectomies. The ridge of Passavant, originally described in 1863, is a transverse fold of the posterior pharyngeal wall, which develops during speech, swallowing, retching, vomiting, or whenever it is necessary to shut off the nose from the mouth. Barton described it as linear and pencil-sized in the adult, and located a few millimeters below the base of the uvula. It extends across the breadth of the posterior pharyngeal wall. Barton believed that the muscle responsible for this ridge, or fold, is almost exclusively the superior constrictor of the pharynx and plays a most important part in closure of the nasopharynx. A discussion and description of the mechanism of the sphincter of the nasopharynx is well presented. Injury to Passavant's ridge during adenotonsillectomy is responsible for a postoperative nasal voice and escape

of fluids through the nose during swallowing. In this interesting article on a little discussed subject, Barton concluded that the ridge of Passavant is not only a significant physiologic component of the nasopharyngeal sphincter but also plays an important part in articulation and deglutition. He pleaded for more care and consideration in performance of adenoidectomies if postoperative speech and swallowing difficulties are to be avoided.

PHYSIOLOGY.

Fyke and Code[5] studied the normal pharyngeal and upper esophageal pressure changes during the act of deglutition. They used a small electrical transducer in a No. 14 Sawyer gastric tube, and measured pressures at various selected levels. They concluded that the cricopharyngeus muscle is in a constant state of tonic contracture during the resting phase, as evidenced by the constant increased pressure in the segment of pharyngo-esophagus occupied by this muscle. They further observed that during the active phase of swallowing, there is not only a great increase in pharyngeal pressure but also a significant negative pressure in the upper esophagus. This combination results in a sharp pressure gradient between the pharynx and esophagus and helps explain the rapid passage of the bolus of food in this area. In addition, during swallowing, there is a sharp decrease in pressure at the cricopharyngeus level; this indicates that the sphincter opens. The pressure then increases at a time that would indicate that the sphincter closes as an integral part of the progressive pressure wave which continues down the esophagus as the peristaltic wave.

In discussing the current evaluation of nasopharyngeal defense mechanism Fenton[6] stated that the first line of defense is the covering of mucous secretion on the epithelium, as produced by the goblet cells and glands of the mucous membrane. Inert dust particles and bacteria are removed with the mucus by ciliary action. The main element in mucosal defense consists, therefore, in a carpet of mucosal cells lining the nose, nasal cavities, nasopharynx and upper respiratory tract. Ciliary action in the nasopharynx and accessory

sinuses provides a fresh sheet of protective mucous secretion over the epithelial surface. Increased mucous secretion to cope with irritation or infection is an attempt to strengthen the first line of defense. The second line of defense of neutrophiles and of "scavenger cells," the histiocytes, which proliferate or migrate and increase rapidly in number, is the early stage of infection of the sinus membrane. The third line of defense is lymphatic drainage, first to the retropharyngeal and superior cervical lymph nodes and eventually into the cervical lymph ducts. Lymphatic drainage is further discussed. This article on an interesting subject is most thought provoking.

ANOMALIES.

Bizarre pain in the neck or pharynx in a previously tonsillectomized patient may sometimes be attributable to a prolonged styloid process, according to Hill[7]. Scar tissue over the process, involving the Vth or IXth cranial nerves, or direct pressure by the process upon the carotid artery may be the cause of symptoms. Diagnosis is aided by roentgenography. Treatment is surgical excision through either the pharyngeal approach of Eagle or the external approach of Soesser and Caldwell in selected cases.

DIAGNOSIS.

Hoover[8] presented a general discussion of several aspects of the interrelationship between surgery of the pharynx and upper esophagus, and the esophagoscopist as a consultant. He discussed the roentgenologic diagnosis of upper esophageai lesions, in which he stressed preoperative lateral roentgenograms of the cervical spine to rule out anterior spurs encroaching upon the esophageal lumen. In regard to esophagoscopy, he mentioned inflation of the esophagus to help bring out the presence of varices. He stressed that multiple lesions must be kept in mind, and carcinoma must be ruled out in all patients older than 30 years.

A unique technique for retrograde esophageal dilatation was described and illustrated. The essential feature of this technique is introduction of multiple small bougies through

the stricture individually, then withdrawal of them simultaneously through the stricture as the esophagoscope is being removed. Their multiple diameters dilate to any desired degree. A lengthy, well illustrated discussion of pulsion diverticula of the upper esophagus, and their relationship to the cricopharyngeus muscle, was given. Hoover described a technique whereby excess pharyngeal mucosa may be pleated or removed in a patient who had a lesion that resulted in pharyngeal dilatation. This, Hoover called "tailoring" the pharynx in order to make it funnel-shaped.

A relatively simple inexpensive device for rapid filming of swallowing, designed as an accessory to the usual screening apparatus of standard fluoroscopic units, was constructed by Walters and Williams[9]; it is operated manually. A photograph of the device, a diagram of its working parts, and reproductions of roentgenograms taken with it are included. It takes ten consecutive films of the pharynx as the patient is swallowing barium.

DISEASES.

Huebner and associates[10] reminded us that the undifferentiated respiratory diseases, the febrile catarrhs and the afebrile common colds continue as unsolved omnipresent sources of human distress. They undertook an extensive study to discover the causative factors, and after much work they reported that new respiratory system viruses have been isolated in tissue culture from adenoids and tonsils surgically removed, and from nasopharyngeal and conjunctival secretions and feces of persons with respiratory illnesses. These new viruses have been designated as adenoidal-pharyngeal-conjunctival agents, indicating the important anatomic sites where they occur. Six types of viruses were recognized, all of which cause frequent infections in human beings, beginning at an early age. The amount and specific kinds of illness produced by these infections are being intensively investigated. Types III and IV cause specific respiratory illnesses. This is the kind of article that must be read in its entirety to be fully appreciated.

In an illustrated article primarily for the general practi-

tioner Ireland[11] attempted to correlate the pathologic findings in nasopharyngeal diseases with the clinical picture. Diseases discussed include polyps, imperforate choana, adenoids, Thornwaldt's cyst, benign fibroadenoma and malignant tumors. He pointed out the potential danger which relatively mild symptoms may indicate.

Catanzaro and associates[12] presented a statistical study of the effect of terramycin on non-suppurative complications in patients with Group-A streptococcal pharyngitis. The study included 986 proved cases; 506 of these were treated and 480 untreated patients, the latter serving as controls. The results were compared with similar cases in which penicillin, chloramphenicol and erythromycin "R" were used. It was concluded that the incidence of subsequent rheumatic fever in treated patients is lower than in the untreated ones. It was further concluded that the drug of choice is penicillin, given in effective concentrations for ten days, and that the other drugs studied should be reserved for patients who cannot take penicillin.

Nuessle, Wright and Jones[13] conducted a study of streptococcus cultures of the throat in 290 hospitalized military men suspected of having a streptococcal infection. Results of this study indicate a higher incidence of positive cultures when sheep blood agar was used than when human blood agar was used as a culture medium. The authors stated that the presence of a factor in human blood, which could theoretically be antistreptolysin, may inhibit growth of streptococcus pyogenes.

Ten to 15 per cent of patients seen by the otolaryngologist complain of postnasal drip, according to Richards[14], who presented a most interesting and unique discussion on this vexing problem. These patients complain chiefly of a sensation of the presence of something abnormal, localized approximately on the posterior superior surface of the soft palate. A desire to get rid of this object is quickly generated. Further symptoms and sensations are described at length. Although the patient is conscious of an apparent mass of mucus, the examining laryngologist seldom finds anything to substantiate this. The film of mucus usually present in the

nasopharynx and pharynx is duly considered. Sinusitis, allergy and atrophic rhinitis seldom account for the discomfort produced by the postnasal drip. Nasal physiology, cytology, sinus infections, allergy and structural abnormalities are thoroughly discussed. It is admitted that excessive amounts of nasal secretions usually represent a transitory response to some external environmental irritant, such as smoke fumes, chemical vapors and the like. Richards concluded that the wide range of local therapy utilized by the modern rhinologist in the office treatment of postnasal drip serves only to emphasize the lack of any really dependable remedy. This article is characteristic of the versatility of the writer, and carries the assurance of being enjoyed by the reader.

A study of a new pathologic entity was presented by Bell and associates[15]. Working in the National Microbiological Institute at Bethesda, Md., in cooperation with local physicians in the vicinity, they studied a specific virus, called the adenoidal-pharyngeal-conjunctival (APC) Type III, which is the etiologic agent in a specific pathologic entity affecting principally children. They suggested that this disease be named pharyngo-conjunctival fever. It appears to be an acute infectious disease transmitted directly from individuals infected, or through the medium of contaminated swimming pools. It is characterized by high fever lasting up to six days, pharyngitis and conjunctivitis. These symptoms may occur alone or in any combination. A specific antibody response results from the infection.

In a symposium on postnasal discharge in which Proetz, Boies and Morrison[16] participated, many interesting facts were brought out. A certain amount of discharge from the postnasal space is considered normal. The nasal mucosa throws off approximately one liter of water a day; less than half of this is consumed in moistening air and the other half slips down the nasopharynx, pharynx and esophagus unnoticed unless the secretion becomes too excessive, viscous, malodorous or obstructive. These latter conditions produce a consciousness of secretion in the nasopharynx and pharynx, which is referred to as postnasal discharge. Some of the

various factors responsible for production of this annoying symptom, such as dry central heating, tobacco, alcohol, dust and fumes, allergies, fungous irritations, atrophy, metabolic and endocrine disturbances and prolonged medication, were discussed. Because postnasal discharge represents one of the commonest complaints of patients seen by otolaryngologists, all practicing this specialty should read this article, which is probably the best on this subject to come under our observation.

Neistadt[17] reported results of a study of a new sympathomimetic agent, tetrahydrosoline hydrochloride, as a nasal decongestant solution (0.1 per cent) in 204 patients (12 with allergic rhinitis and 192 with nasopharyngitis or sinusitis or both). All but one patient, whose response was only partial, reported excellent results. In addition to its promptness of action, degree of relief provided and percentage of patients benefited, it displayed prolonged action and no untoward local or systemic effects.

Weinstein and Sachs[18] reported a case of a 70-year-old woman with pemphigus of the mouth, oropharynx and larynx without cutaneous manifestations. Since primary pemphigus involving only these areas is uncommon, the case is described in detail. Because of laryngeal stenosis caused by the pemphigus, tracheotomy was required. These authors briefly reviewed the literature and discussed the micropathology of the lesion.

Hoover[19] discussed his general observations and experiences at the Lahey Clinic on the functional abnormalities of the pharynx and their sequelae. He described dysphagia due to vagus nerve paralysis and illustrated this with case reports. This leads into the subject of pulsion diverticula of the pharyngoesophagus and the accompanying spasm or stenosis of the cricopharyngeus. Cricopharyngeus relaxing procedures and plastic reshaping of the hypopharynx are described in the case of vagus paralysis, as well as pulsion diverticulum. Unlike Lahey, who preferred the two-stage diverticulectomy, Hoover considered the single stage procedure preferable. The latter portion of this article deals with general considerations of esophagoscopy.

Lindsay[20] reviewed the various neurologic, psychologic and structural disturbances that might cause functional difficulties of the upper swallowing mechanism. In a general discussion of the swallowing mechanism, he pointed out that its direct action cannot be observed, but fluoroscopy and roentgenography can provide useful objective evidence. Disturbances due to poor muscular action, interrupted innervation, and psychomotor influences are discussed. The article is illustrated with appropriate roentgenograms. Roentgenography can confirm weakness or asymmetry of action of the constrictor muscles and of the cricopharyngeus commonly seen in disturbances of musculature and innervation, but present techniques cannot demonstrate evidence of increased tone or spasm of these muscles.

Benedict and Sweet[21] reported two cases of dysphagia due to hypertrophy of the cricopharyngeus muscle or hypopharyngeal bar. They quoted Templeton and Kreider, who stated that the cricopharyngeus muscle is a true sphincter which can be demonstrated roentgenologically. When the Valsalva test is performed, contraction of the cricopharyngeus is intensified. Brunner and Sweet stated that the purpose of the tonic contraction of the cricopharyngeal sphincter is to prevent esophageal respiration. Relaxation of the cricopharyngeal sphincter would cause air to be sucked more readily into the esophagus than into the lungs. The mound on the posterior pharyngeal wall at the level of the cricopharyngeus muscle may be called the "hypopharyngeal bar," and this indicates spastic contraction of the cricopharyngeal muscle. In the two cases of dysphagia, reported by Benedict and Sweet, hypertrophy of the cricopharyngeus muscle was demonstrated roentgenologically, and since all other causes of dysphagia were eliminated, it was concluded that the presence of a hypopharyngeal bar was responsible for the existing dysphagia.

DIVERTICULA.

A good short general review of diverticula of the upper esophagus, without author credit or references, appeared in the *Medical Times*[22]. No cases or statistics were included.

In a brief but excellent article Neville[23] cautioned that although most pharyngo-esophageal diverticula can easily be handled through a cervical approach, occasionally one may encounter an extremely large diverticulum projecting into the thorax. In such cases it may be judicious to open the thorax and free the diverticulum from continuous intrathoracic structures, after which the usual surgical approach can be employed and the diverticulum easily lifted into the neck. Such a case was encountered by Neville. Because practically all pharyngoesophageal diverticula measure less than 5 cm., most of them can be removed without difficulty through the usual cervical incision; however, considerable difficulty may be experienced in attempting to remove extremely large ones through such an incision because of the proximity of major vessels and the possibility of injury to these structures. The thoracic approach minimizes these dangers by freeing the diverticulum from the proximity of these structures.

In a discussion of pharyngo-esophageal diverticulectomy Jackson and Norris[24] called attention to the fact that approximately 75 per cent of their cases occurred in males, but offered no explanation for this. Although it is generally accepted that pharyngo-esophageal diverticula occur only in elderly people, two cases are reported in individuals 21 and 27 years of age. The surgical technique employed is identical with that described by other surgeons, and in keeping with the present day trend, they preferred the one stage technique. Esophagoscopic guidance in determining amputation of the neck of the diverticulum is advocated. The one complication of stenosis in their series occurred in a patient in whom esophagoscopic guidance was not used. Having experienced the same complication under identical conditions as reported by Jackson and Norris, we heartily concur that esophagoscopic guidance is highly desirable during amputation of the neck of the diverticulum.

A brief review of the problem of pulsion diverticulum of the upper esophagus is offered by Howden[25]. He presented reports of nine cases in which single-stage diverticulectomy was performed and gave a detailed outline of the surgical technique used.

Johnson[26] believed that the otolaryngologist is unnecessarily depriving himself of a stimulating field of surgery, namely, repair of pulsion diverticulum of the hypopharynx. He stated that most recent references to this lesion are found in the general surgical literature, despite the fact that the anatomic and surgical features clearly fall within the practice of the otolaryngologist, who is interested in surgery of the neck. Johnston covered the subject well in a short discussion, and included a case report.

Fox and associates[27] apparently considered pharyngeal diverticulectomy a procedure for a relatively uncommon condition. They presented the first such case seen at the Kent General Hospital. Nothing new or constructive is offered in reporting a routine case. It is surprising to read that a stump remained after excision of the diverticulum and that the opening into the hypopharynx was closed with suture material as large as chromic No. 1. This, coupled with the fact that phenol and alcohol were applied to the stump, is reminiscent of a general surgeon performing an appendectomy. The authors reported that the diverticulum developed on the lateral wall of the hypopharynx, whereas it has been established that all such diverticula invariably develop in the median line of the posterior wall and gradually develop to one side or the other, most frequently to the left side.

Treatment of traumatic perforations of the esophagus has undergone considerable change in the last two decades. Prior to 1935 uncontrolled mediastinitis and death followed esophageal perforations in 70 per cent of the cases unless surgical drainage was instituted. Kesseler and Maier[28] stated that early recognition and prompt treatment of traumatic perforations of the esophagus are essential to preserve life. Although the early use of antibiotics may lower the mortality rate considerably, immediate surgical intervention is usually necessary. Kesseler and Maier reported a case of perforation in the lower portion of a diverticulum. Surgical drainage was instituted by performing right cervical mediastinotomy; however, the exact location of the perforation was not initially known. When temperature persisted, a search revealed the location of the perforation and diverticulectomy was performed with beneficial results.

ROENTGENTHERAPY.

Reich[29] emphasized the fact that radiologists have long recognized that lymphoid tissue is the most sensitive of normal structures in the human body, to radiation. Recognizing the value of the Crowe radium applicator as used in the nasopharynx, and its limitations, Reich conducted a study of 105 patients referred for roentgentherapy to the nasopharynx. Of these 68 were available for follow-up study. The ages of the patients ranged from 18 months to 55 years. Thirty-eight with hypertropied lymphoid tissue in the nasopharynx complained of loss of hearing. After treatment 28 patients showed improvement. Good results were obtained in five patients with soft tissue masses in the nasopharynx treated by radiation. Other types of cases were treated successfully so that Reich was forced to the conclusion that roentgentherapy in small doses is a useful adjunct for treating hypertrophied lymphoid tissues in the nasopharynx, especially if the presenting symptom is deafness.

Green and associates[30] presented an excellent discussion of the indications for and technique of irradiation of the Eustachian tubes for the reduction of lymphoid tissue that is blocking adequate drainage of the middle ear. They sent questionnaires to the parents of 165 children treated by external Roentgen-ray irradiation during a period of five-and-one-half years. The answers received would indicate less hearing loss, less mouth breathing and snoring, and less respiratory infections following Roentgen therapy. It should be noted, however, that no control group of untreated patients was included for comparison.

An editorial[31] in the *British Medical Journal* reviewed the works of Crowe, Guild, Bordley and Hardy on the effects upon hearing defects of radium therapy to the nasopharynx of children. The hope was expressed that further investigations on this subject might follow.

Van Herik and Erich[32] devised a bakelite applicator for radium therapy to the nasopharynx. The technique for use of the applicator was described and well illustrated. They used this technique to supplement Roentgen therapy for

squamous cell carcinoma of the nasopharynx in 30 patients without untoward complications.

SURGERY.

A number of aspects of the subject of tonsillectomy was discussed by Moore[33], in the light of his experience. He reviewed the subject of tonsillectomy with use of a general anesthetic, and described his technique of peritonsillar injection of a vasoconstricting solution to facilitate the procedure and to limit loss of blood. There was a brief discussion and warning about the use of efocaine "R" for prolonged relief of pain following tonsillectomy. Moore concluded with a review of possible untoward and possible beneficial effects of ACTH and Cortisone as related to the problem of tonsillectomy performed with use of a general anesthetic. This is a timely and interesting article on an old subject.

Marshall and associates[34] were favorably impressed with Kutapressin, a non-toxic, selective vasoconstrictor, in the prevention of hemorrhage following tonsillectomy and adenoidectomy. No scientific information concerning the chemical or pharmacologic nature of the product was furnished, nor did they mention animal experiments to determine possible toxic qualities of the drug. No control series of cases was presented, and no factual information was given concerning the 100 cases mentioned.

A questionnaire was mailed by Hamrick[35] to every hospital in the United States approved for residency training in otolaryngology and anesthesiology in an effort to study anesthetic preferences for tonsil and adenoid operations in children. The results from 155 hospitals were tabulated. The first choice was vinyl ether induction with ether maintenance by insufflation. Hamrick listed the first ten choices and stated that endotracheal techniques are increasing in popularity, owing to their well recognized margin of safety in the hands of the well trained anesthetist.

A study of the effects of depot local anesthetics injected into the operated area after tonsillectomy was made by Reed and Montgomery[36] at the Massachusetts Eye and Ear Infirm-

ary. They used efocaine in their study. Of the 90 patients, only 27 received the drug; the remaining 63 served as controls. They concluded that this drug has no significant effect upon the severity or duration of the postoperative discomfort or complications.

Folds and pockets in the nasopharynx are observed surprisingly often in routine nasopharyngeal examinations. Tolczynski's[37] investigations revealed their incidence to be 47.5 per cent, and the nasopharynx presented a variety of strings, folds, bands, crypts and pockets which varied with each patient. Each of these is illustrated and described in detail. Tolczynski stated that the residual masses, after a faultily performed adenoidectomy, are more susceptible to infection than the intact adenoid. Deep folds and pockets in the nasopharynx are largely responsible for annoying postnasal drip due to chronic infection. Adenoidectomy under direct vision with use of a general anesthetic is the method of choice in the prevention of formation of folds, crypts and pockets. This is an interesting and illuminating article that should be read by all otolaryngologists performing adenoidectomies.

BENIGN TUMORS.

Surgical removal of benign nasopharyngeal tumors frequently taxes the ingenuity of the surgeon. The inaccessibility of the nasopharynx and the vascularity of the lesions arising in that area are factors which must be overcome for successful management of these tumors. White[38] pointed out that the principal benign tumors found in this area are nasopharyngeal fibromas and large choanal polyps. Removal of the former type presents a formidable problem at times because of severe bleeding. White described various approaches and techniques advocated for removal of these tumors, and described a combined intranasal and postnasal approach that he devised and used successfully for removal of fibromas. It consists in passing a loop of wire through the nose into the oropharynx, and with appropriate palate retractors, placing the wire loop around the tumor. An insulated rigid tube protects the nasal mucosa while a cautery tip makes contact

with the wire. Pulling down on the wire loop resects the tumor as closely to its base as possible with minimum bleeding.

Mills[39] reported a case of midline cyst of the nasopharynx which could be seen protruding a few millimeters below the level of the uvula. A preliminary diagnosis of polyp was made, and a transpalatal approach was used to expose the cyst to better vision. The dome of the cyst was excised by diathermy and a small blind pit was exposed. It was concluded that this cyst was formed from the vestigial remnants of Rathke's pouch.

We are reminded by Beales[40] that simple tumors of the pharynx are uncommon. Probably the commonest of these is the "mixed salivary tumor." It arises from the three pairs of salivary glands, as well as from salivary gland tissue regardless of its location. Thus, these tumors arise from the small glands that are widely situated throughout the oral cavity in the lips, cheek, palate and floor of the mouth. The serous cell is probably more prone to metaplasia than the mucous type; therefore, salivary tumors are commoner in the parotid gland. Beales reported an interesting case diagnosed as a peritonsillar abscess which proved eventually to be a "mixed salivary tumor." An attempt was made to remove the tumor intra-orally; but the futility of this approach was quickly realized, and the operation was terminated. Later through an external approach the tumor was removed without difficulty. The unsuccessful original operation emphasizes the fact that sizable tumors should never be approached intra-orally, as they are not only difficult to dissect, but control of hemostasis may become such a problem as to place the patient's life in jeopardy.

Two interesting and unusual cases of plasmacytoma originating in the nasopharynx are presented by Emslie-Smith and co-workers[41]. In both cases the initial lesion was a soft tissue plasmacytoma of the nasopharynx and adjoining areas, followed by appearance of widespread cutaneous plasmacytoma and involvement of bone and lymph nodes. Extramedullary plasmacytomas are uncommon tumors, affecting males more frequently and generally occurring in the mucous membranes of the upper air passages. Their histologic appearance gives

little clue to their future course which varies from complete recovery with treatment to the development of myelomatous lesions throughout. As a rule these lesions respond well to irradiation early in the illness, but later are more resistant.

Mixed tumors in the orofacial region are common, but mixed tumors located specifically on the posterior pharyngeal wall are rare. Havens and Butler[42] reviewed 41 cases encountered at the Mayo Clinic among 1,500,000 clinic registrations during a period of 37 years. After reviewing the general subject of mixed tumors and commenting on methods of treatment in the past, they discussed the surgical approaches used in their 41 cases. They believed that an external approach is advisable in some cases of pharyngeal mixed tumor. Their follow-up has been good and their recurrence rate low. They observed that there seemed to be less "seeding" in the wound after internal excision than after the external incision. They believed that this might be explained by the fact "that a clean surgical wound that is closed, as a neck wound is closed, would provide much better soil for possible 'seeding' than would a wound which is packed wide open with iodoform gauze and which is left in place for four to seven days."

Epstein and Loeb[43] reported an unusual case, in an infant three-weeks-old, in whom a thymic tumor was found in the hypopharynx. The original discovery was made by the baby's mother, who noted what appeared to be a second tongue when the infant regurgitated. No difficulty was encountered in its removal and the pathologist rendered a report of hamartoma of the pharynx. Epstein and Loeb reminded us that the presence of an aberrant thymus above the level of the thyroid gland is rare. After extensive study they concluded that this thymic tumor was probably of embryologic origin from the fourth visceral cleft.

A general review of juvenile fibromas of the nasopharynx by Erich[44] emphasized the dangers involved in biopsy of these lesions. Erich believed that biopsy should not be performed if the diagnosis is obvious clinically, and if biopsy is necessary it should be done only in the hospital where adequate precaution can be taken to control hemorrhage. The pathology, etiologic factors, symptoms and treatment were re-

viewed. Erich's present choice of therapy is implantation of radon seeds repeatedly every three to six months until the tumor has disappeared. The specific technique for radon implantation was described.

MALIGNANT TUMORS.

Duboe[45] stressed the importance of examination of the nasopharynx of each new patient coming to the office, particularly if the patient's symptoms suggest nasopharyngeal disease. He properly emphasized the fact that practically every nasopharynx can be examined adequately if the examiner is willing to take sufficient time to carry out this examination. Malignant tumors of the nasopharynx are more common in this area than are benign tumors, and the usual sites for these malignant lesions are the fossa of Rosenmuller, posterior wall on either side of the midline and roof of the nasopharynx. Nasopharyngeal cancer is relatively common in China, and in Hong Kong it is reported to be the second commonest malignant disease. The prognosis is unfavorable even though the lesion is discovered early. Sarcomas offer a more favorable outlook than do carcinomas. Malignant lymphoma, reticulum cell sarcoma, lympho-epithelioma, and transitional cell carcinoma respond well to irradiation, but the rate of recurrence is high. Involvement of the cranial nerves makes the prognosis more unfavorable and metastasis is usually limited to the cervical nodes.

In an excellent presentation Missal[46] discussed the problem of adequate surgical treatment of extensive carcinoma of the pharynx, larynx and cervical esophagus, which he considered to be one of the most distressing clinical problems encountered by the laryngologist. In the past most of these neoplasms have been considered inoperable and irradiation has been the principal therapy offered. The benefits achieved in these cases by irradiation leave much to be desired. For this reason some of those lesions are now being excised with encouraging results. Missal did not use the term radical surgery in these cases but preferred using the designations of adequate and inadequate surgery. Use of these terms is predicated upon the existence of three factors: first, the surgeon must possess

sufficient skill and knowledge to perform an adequate operation; second, he must execute the required surgical procedure, and third, he must exercise sound judgment in the selection of patients suitable for the operation. A historical review, as well as critical comments on cases presented, adds to the interest of the presentation. Surgical technique is discussed and emphasis is placed on preoperative preparation of the patient with proper attention to fluid and electrolyte balance. Although this presentation includes the pharynx, it is principally concerned with surgical treatment of malignant lesions of the larynx, pyriform fossa, mouth of the esophagus and with neck dissection. Since the present review encompassses only pharyngeal surgery, this most excellent presentation is briefly sketched and those interested should read the entire article.

In a review of the treatment of hypopharyngeal carcinomas Morfit[47] advocated laryngopharyngectomy and simultaneous block dissection of the neck as the treatment of choice. He concluded that other forms of therapy have not yielded good statistical cure rates, and believed that wide dissection would improve the incidence of cures. His discussion included an illustrated operative description and details of postoperative problems. No case reports or statistical studies were offered in support of the opinions expressed.

Jack[48] discussed the surgical problem that carcinoma of the hypopharynx and upper esophagus presents. He believed that wide resection of the tumor with immediate reconstruction of the area and restoration of swallowing should be the desired objective. He reported the case of a postcricoid lesion with involvement of the esophagus from the level of the cricoid for a distance of about 6 cm. Left postero-lateral thoracotomy through the bed of the resected eighth rib offered an approach to the esophagus and stomach. After the operation was completed in this area, the patient was placed on his back and laryngo-pharyngectomy and partial esophagectomy were performed. The lower portions of the esophagus and stomach were brought up and the upper portion of the esophagus was sutured to the pharynx (pharyngo-esophageal anastomosis). The author described an interesting and formidable surgical procedure, but failed to include follow-up notes or discuss prognosis.

Martens[49] reported what he believed to be the first recorded case of malignant synovioma in the hypopharynx. A photograph of the gross specimen and a diagrammatic drawing were shown. Five cases of benign and malignant synovial tumors from other regions of the body were also described, and the general pathology was briefly discussed.

Green[50] discussed the mutual invasion of two independent tumors occurring simultaneously in the same pharynx. One was a squamous cell carcinoma of a tonsil, and the other a fibrosarcoma of the lateral pharyngeal wall. Where the two had been in opposition to each other, mutual implants had occurred at the site of collision. This paper was written by a pathologist and is well documented by photomicrographs of the tumors.

Sanghvi and coworkers[51] conducted an investigation on the smoking and chewing of tobacco in relation to cancer of the upper alimentary tract. Their studies showed that intraoral cancer is relatively more prevalent in Bombay than in New York or London. Even in regard to the commonest site of oral cancer there is a noticeable difference in various localities in India. Cancer of the lip is the commonest type of cancer in Bihar; of the buccal mucosa and alveolar margin in Travancore and some parts of Madras; of the base of the tongue in Bombay; of the oral surface of the palate in Andhra, and of the hypopharynx in Assam. Personal history and habits relative to alcohol, smoking and chewing were studied carefully, and the results of their investigation showed that the habit of chewing was associated with cancer of the oral cavity; that the combined habit of smoking and chewing was associated with cancer of the hypopharynx and base of the tongue; and that smoking alone was associated with cancer of the oropharynx and esophagus.

The surgical treatment of extensive cancer of the cervical esophagus, pharynx and larynx, when it has been necessary to remove the total circumference of the swallowing tube, has been encumbered by the delayed multiple stage reconstruction of the pharynx and esophagus by use of transposed tissue flaps from the neck or chest. Conley[52] described an operation designed to combine the excisional and reconstructive stages

into a single composite one stage procedure. After radical resection of the cancerous growth, the continuity of the pharynx and cervical esophagus is restored by implantation of a thick, split free skin graft draped over a woven nylon tubular stent which is carefully sutured to the mucous membrane. Conley described and illustrated the technique for this all-important surgical procedure, which undoubtedly represents the latest advance in reconstructive surgery for such cases.

The twelfth reported case of carcinoma occurring in a pharyngeal or esophageal diverticulum was presented by Riberi, Battersby and Vellios[53]. They stressed the diagnostic importance of a sudden loss of weight and a change in character and severity of dysphagia in long standing cases of pharyngoesophageal diverticula, as evidence of possible malignant lesions. They successfully treated their patient, a woman 82 years old, by surgical excision of the diverticulum with its carcinoma, and they advocated this therapy for all such cases in which the malignant growth is still localized to the sac.

Batsakis and associates[54] reported a case of nasopharyngeal angiofibroma containing in its center a fibrosarcoma. The patient was 54 years of age at the the time of definitive surgical removal of the tumor and was 48 years of age at the time of the original diagnosis of angiofibroma. Several attempts at removal were made during this five year period, and extensive radiation therapy was then given. The tumor continued to be considered benign pathologically until after radiation therapy. Final excision 11 months following irradiation produced a specimen in which the malignant changes were found. The relation of these changes to "radiation cancer" was discussed briefly.

The origin of nasopharyngeal chordoma was briefly described by Crikelair and McDonald[55]. A nasopharyngeal chordoma is a slowly growing tumor that invades and destroys locally. Radiation therapy will result in a temporary favorable response in some cases and subtotal removal of large tumors may prolong life by relieving compression. A case is reported of a white boy in whom symptoms referable to a nasopharyngeal tumor developed at the age of 11 years.

Radiation therapy, radium needles and surgical treatment have made it possible to keep this patient alive for eight years (at the time of the report the patient was 19 years old). The accompanying photographs show massive facial distortion before treatment and marvelous results following plastic surgery.

Lawley and Mekie[56] studied the effects of urethane and triethylene melamine (TEM) on separate series of patients with nasopharyngeal malignant lesions. Temporary palliative effects were noted in 12 of 16 patients who completed a full course of treatment with urethane. They believed that the possibility of using urethane as a form of palliative therapy should be considered when: *a*, radiotherapy is not available; *b*, the disease is too advanced for radiotherapy; and *c*, there is an interval before radiotherapy can be begun. No significant improvement could be attributed to TEM in the seven patients treated. Leukopenia and pronounced fall in the number of granulocytes developed after administration of the drug.

St. John and Woo[57] commented on the rarity of rhabdomyosarcomas, and further pointed out that these tumors in the nasopharynx occur chiefly in childhood or adolescence, and at first have the appearance of nodular polypoid tumors which are white or flesh colored, depending upon the amount of vascularization. They reported such a case in a four-year-old boy. The nasal passage was completely filled with a polypoid-like tumor. A mass could be seen protruding from behind the uvula. This polypoid tissue was removed by means of a nasal snare, and a diagnosis of rhabdomyosarcoma was established. Roentgen therapy controlled growth of the tumor only temporarily.

A case of dilatation of the pharyngo-esophagus following total laryngectomy was reported by Cracovaner and Rubenstein[58]. The dilatation was so great that it interfered with deglutition and esophageal speech. They discussed possible mechanisms of formation and described a method of repair.

Ashley, Briggs and Affley[59] reviewed the histories of four of their patients requiring reconstructive surgery of the

pharynx following radical excision for carcinoma. Each case illustrates a different technique and type of repair. Photographic and diagrammatic illustrations greatly aid the descriptive text. Of special interest is the first case, in which primary closure of a strip of remaining pharyngeal mucosa was accomplished over a gastric feeding tube. Then in two days the gastric tube was removed, and a feeding gastrostomy was performed. Liquids were begun by mouth 18 days postoperatively. No details were given concerning the rationale for removing the nasogastric tube and maintaining nutrition through a gastrostomy.

A clear concise discussion of wide surgical excision of carcinoma of the hypopharynx was offered by Snitman and Horwitz[60] as a preliminary report. An implied promise of follow-up studies, as well as a definite statement that a paper is being prepared concerning reconstructive phases of the work, adds to the depth of the paper. These authors classified carcinomas of the hypopharynx into three groups, depending upon gross spread of the primary lesion. They believed that operation is indicated in all cases in which the malignant tissue can be removed *en bloc*. They considered reconstructive techniques to be adequate to make even complete excision of the pharyngeal mucosa feasible, even though no pharyngeal mucosa be left to close or to participate in future closure. Their early cure rate has been better than in any other type of therapy. A five-year analysis is not yet available.

Moore and Martin[61] reviewed all cases of primary melanoma of the oral cavity and upper respiratory tract that occurred at the Memorial Center in New York between 1930 and 1948. They found records of 26 cases that they considered acceptable. There were four cases of melanoma of the oropharynx; all were limited to the palatine tonsil. Only one patient had initial symptoms related to the primary site, whereas the other three had initial symptoms from metastatic lesions. Two of the four primary lesions were non-pigmented. None of the patients was given therapy directed toward a cure. Melanomas in other areas, such as the larynx and sinuses, were included.

After discouraging results with other methods of pharyngeal and upper esophageal reconstruction, Wilkins[62] began to employ a new method, utilizing the laryngeal and upper tracheal mucous membrane with the anterior portion of the laryngeal cartilages. He dissected the mucosa off the anterior face of the posterior half of the cricoid, if the carcinoma were postcricoid, and used this mucosa for reconstruction. He believed that the barrier of the cricoid cartilage should be adequate to prevent involvement of this anterior mucosa by the disease. The technique as used in two cases, was described and illustrated in detail. This article is recommended reading for all otolaryngologists interested in cancer surgery of the neck.

Rosenbaum and Seaman[63] called attention to the general failure to appreciate the neurologic manifestations associated with nasopharyngeal tumors. Their statement that intracranial extension is the earliest presenting sign of nasopharyngeal malignant tumors can easily be challenged. The anatomic location of the tumor favors early intracranial extension and neurologic signs particularly because of the proximity of the foramen lacerum and ovale and the IIIrd, IVth, Vth and VIth cranial nerves. The commonest initial symptom produced by these tumors, steady unilateral facial or aural pain, should demand thorough nasopharyngeal examination. Obstruction of the Eustachian orifice, blood streaked sputum or cervical adenitis are some of the other presenting symptoms. As the tumor grows larger, it will produce neurologic symptoms by invasion of cranial nerves. Unfortunately, however, in our experiences, tumors in this area are frequently not diagnosed early because they produce few symptoms in the early stages. We have found neurologic symptoms as late indications of tumors of the nasopharynx.

In a general discussion, designed for general physicians, Sebai[64] discussed trends in treatment of cancer of the lip, tongue, oral cavity, pharynx, tonsils and larynx. He concluded that the trend is toward more and earlier radical excision, and that this attitude is becoming noticeable even in countries, such as Sweden and France, where irradiation therapy has generally had its best results. The reasons for

this apparent acceptance of radical excision are the advances in anesthesiology, antibiotics, blood banks and plastic repair. These advances permit more meticulous and time consuming surgical procedures than was formerly possible, and the cure rate has therefore been better. The figures of the Memorial Cancer Hospital of New York during the past ten years strongly substantiate this tendency. Several pages of photographs of the author's patients, who had undergone radical excision in the bucco-pharyngeal area, are included in an extremely readable paper.

In an excellent article on the indications and technique for cervical node resection Ochsner[65] discussed at length the problems confronting the surgeon when dealing with carcinomas of the lip, gingiva, tongue, floor of the mouth, soft palate, tonsil and pharynx. In the discussion of each of these the advisability of radical neck dissection was given due consideration; prophylactic radical resection was also discussed. Carcinoma of the tonsil represents about 10 per cent of all buccal cancers and involvement of lymph nodes is frequent and early. The treatment advocated for carcinoma of tonsils consists of intense irradiation followed by *en bloc* excision of the original lesion and unilateral radical neck dissection. This offers the patient the best chance of survival. Cancer of the nasopharynx is usually a highly malignant tumor and may be either epithelial or mesothelial in origin. Lymph nodes become involved early because of the rich lymphatic drainage in the region of the nasopharynx. Treatment consists in intensive radiation. Ochsner then described his technique of radical lymph node dissection of the neck. This is a most excellent article which should be read by all doing this particlar type of work. Lymphatic drainage from the various malignant areas is clearly and adequately illustrated, showing the location and the frequency of involvement of the various lymph nodes of the neck.

In an interesting and extremely readable paper on a subject that is receiving increasing attention, Hadfield[66] reported his methods and results in 24 surgically treated patients with cancer of the nasopharynx. His main concern is with successful and early closure of the pharynx, and he discussed

the Wookey repair and several modifications. His best results were obtained by use of a combination pedicle graft and Thiersch graft, followed by secondary closure. Those readers interested in this subject should read the article and study the diagrams in order to appreciate fully the thoughts expressed.

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MEDIASTINAL EMPHYSEMA COMPLICATING TRACHEOTOMY AND THYROIDECTOMY.*

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Mediastinal emphysema, or pneumomediastinum, is defined as the presence of air in the mediastinum. Although not common, its occurrence as a complication of operations about the neck is frequent enough to warrant an alert awareness of the possibility of its development in these situations. A thorough understanding of the mechanism of its production and an adequate appreciation of its clinical significance is essential for the intelligent management of these cases. The early recognition of its presence and the institution of prompt and appropriate measures to control it are important, and may be life-saving.

Air may gain access to the mediastinum by several anatomic routes:

1. Directly through a perforation of the trachea, bronchi, or esophagus.

2. By dissection along the fascial planes of the neck.

3. From the interstitial tissues of the lung, by travelling to the hilus along the perivascular and peribronchial sheaths following rupture of pulmonary alveoli.

4. From the retroperitoneal space.

It is also theoretically possible for mediastinal emphysema to develop by the spread of air between the parietal pleura and the chest wall, or beneath the visceral pleura, or by passage of air from the pleural cavity to the mediastinum. Finally, artificial pneumomediastium may be deliberately induced by direct needle puncture, as originated for diagnostic purposes by Condorelli[1] in 1936.

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In mediastinal emphysema complicating operations on the neck such as tracheotomy and thyroidectomy, the only pathways requiring consideration are: direct perforation of the airway, spread along the fascial planes of the neck, and through the mechanism of interstitial pulmonary emphysema.

A review of the increasing number of case reports of this complication reveals a great variance in opinion among authors as to the mechanism involved.

Historical Aspects.

Emphysema in that form in which gas or air becomes collected under the skin or in the connective tissue was well known to Paul of Ægina[2] in the Seventh century A. D. There is little doubt that gas gangrene was included in this category, and it was not until the time of Ambroise Paré[3] in the Sixteenth Century that a more accurate concept of aberrent air in the subcutaneous tissues after trauma was developed; however, the principle of negative intrathoracic pressure was unfamiliar to these observers, and no notice was taken of the effect of trapped air within the thorax in patients exhibiting surgical emphysema with chest injuries. In 1767, William Hewson[4] reached the important conclusion that air is contained between the pleural layers in pneumothorax, and he decided it could be the cause of deleterious pressure symptoms even capable of producing death. His interest had been aroused by several reports[5,6,7,8] of chest wounds complicated by subcutaneous emphysema. In the light of the new knowledge gained from his experiments, he reevaluated these cases that had been erroneously interpreted, thereby gaining for them a niche in any history of this subject.

In 1826 Laënnec[9] published the second edition of his classical treatise on diseases of the lung, in which he accurately described the clinical condition of pulmonary interstitial emphysema, so frequently the precursor of mediastinal emphysema. In this great work, he displayed familiarity with certain aspects of its pathogenesis and with many of its symptoms and signs.

The clearer perception of pulmonary disease and the novel methods of physical examination of the chest introduced by

Laënnec stimulated fresh interest in pathology of the lungs. This manifested itself, particularly in France, in a series of reports[10,11,12,13] of clinical cases exhibiting the three stages of pulmonary, mediastinal and subcutaneous emphysema, and the correlation of them all as phases of a single entity termed generalized emphysema.

The observations of these clinicians were based almost entirely on patients with medical diseases associated with severe coughing, or on those with conditions accompanied by intense straining, as in parturition (Ménière's three patients).[10] The appearance of emphysema was readily and satisfactorily explained by Laënnec's "Emphyseme Interlobulaire du Poumon" and its sequelae. From their experience it was believed that this complication in itself was not dangerous, and Laënnec's statement that he had not seen anyone die of this condition alone was cited by Roger[13] in 1862 in support of this conviction ("Je n'ai vu mourir personne de cette affection seule").

Meanwhile surgeons were encountering cases of mediastinal emphysema associated with chest injuries, in which the mechanism appeared to be identical with those of medical origin. Dupuytren[14] regarded the infiltration of air into the mediastinum in traumatic cases with extreme gravity because of the presumed pressure on vital organs, and he attached an unfavorable prognosis to its advent. The whole subject received careful consideration by Demarquay[15] in his monograph published in 1886. He disagreed with Dupuytren's pessimistic view of mediastinal emphysema, because he did not believe sufficient pressure could be built up to affect seriously the important mediastinal organs when an easy escape route was present into the neck and subcutaneous tissues where the emphysema usually reveals itself first. He conceded, however, that in general the complication in surgical cases should be taken seriously, not because of any influence it had on the prognosis but as an indication of the gravity of the primary lesion causing it. He considered spontaneous emphysema, or that following effort, as not serious and as always terminating in cure.

Both Guillot[11] and Ozanam[12] had studied their autopsy material, and Rokitansky[16] completed Laënnec's picture by describing the pathological aspects in his text-book on pathology

in 1856. Following this, in 1888 Müller[17] summarized the course of events from rupture of the pulmonary alveolar walls to interstitial emphysema of the lungs, thence to invasion of the mediastinum with air and finally to infiltration of the subcutaneous tissues of the neck to produce generalized emphysema. In addition, he noted the diminution of the area of cardiac dullness in mediastinal emphysema and described crepitant sounds dependent upon the heart beat. This report exhibited a comprehension of mediastinal emphysema approaching the modern concept.

In pursuing the development of this theory which eventually became known as the intrinsic mechanism, two more recent important contributions must still be given consideration. Through his clinical studies of mediastinal emphysema of apparently spontaneous origin, full credit is due to Hamman[18] for elucidating the diagnostic aspects of interstitial pulmonary emphysema and its sequel, mediastinal emphysema. The pathognomonic sign of air in the mediastinum described as a crunching or crackling sound over the heart synchronous with each contraction has, since his description of it, been known as Hamman's sign.

About 1930, and in subsequent years, the Macklins[19] by their laboratory investigations and voluminous writings greatly increased our knowledge of the pathogenesis of interstitial and mediastinal emphysema. They discussed the sequence of events and the factors involved in the rupture of the marginal alveoli and the transport of air by way of the sheaths of the pulmonary blood vessels to the hilus and thence to the mediastinum; furthermore, they stressed the potential dangers of this aberrent air within the thorax and employed the word "malignant" in describing[19c] these serious cases. The appropriate term "air block" was coined by one[19c] of them to describe the condition arising from interference of air in the lung, mediastinum and extensions therefrom with the vital functions of respiration and circulation.

The drift toward conformity in explaining the mechanism of mediastinal emphysema noted in the Nineteenth Century was halted in 1882 by a scientific inquiry reported by Champneys.[20a] This experimental investigation was conducted in the

true Hunterian tradition, and the conclusions derived from it marked the beginning of a new concept of the manner in which air may reach the mediastinum. The experiments were performed on the bodies of still-born children that were subjected to artificial respiration after inserting a cannula into the trachea following a tracheotomy. Mediastinal emphysema occurred in seven out of 21 experiments, and it appeared that during inspiration the air travelled through the cervical incision into the tissues of the neck and mediastinum. He concluded that the conditions favoring the production of mediastinal emphysema were division of the deep cervical fascia during violent inspiratory efforts resulting from obstruction in the air passages, and that the route selected by the air was the space behind the deep fascia. A second report[20b] in 1884 contributed positive post-mortem findings of mediastinal emphysema in 16 of 28 fatal cases of tracheotomy, presumably all in young children.

Few records of mediastinal emphysema following tracheotomy are available in the literature prior to Champney's publication. Martini's equivocal cases[21] of two infants in 1858 who also had mediastinal abscess, Wilks' three cases[22] (two children and one adult) in 1860 and Bartel's boy[23] of seven in 1866, probably comprise the earliest recorded cases. All these patients died and were examined post-mortem, but no convincing theory of the course of the pathological changes was adduced.

It seems worthy of note that mediastinal emphysema as a complication of tracheotomy was either unrecognized or disregarded for a century or more. Although the operation is credited[24] to Asclepiades in 124 B. C., and was described by many surgical writers of antiquity, the first successful tracheotomy is ascribed to Brasovola[25] in 1546. The procedure was not often performed until advocated by Louis[26] for the removal of foreign bodies in the air passages in his celebrated "Second Memoir on Bronchotomy", published in 1759. Louis noted one fatal case of mediastinal emphysema occurring in a seven-year-old girl with a bean lodged in the trachea, but made no mention of its occurrence following tracheotomy. Similarly in this country Gross,[27] who popularized opening the

trachea for the removal of foreign bodies, appeared to overlook the complication. Among accidents of tracheotomy, he stated that emphysema occasionally attended or followed the operation, but that it was more alarming than dangerous. His subsequent description was obviously that of subcutaneous emphysema of the neck. Weist[28] in 1882 reported on a study of 1000 cases of foreign bodies in the air passages to determine the propriety of bronchotomy in such accidents. The operation was performed in 338 cases with a mortality of 27.4 per cent. Among the 93 fatalities the causes of death were enumerated with no mention of mediastinal emphysema.

During this period one might suppose that mediastinal emphysema complicating tracheotomy for the relief of acute inflammatory obstruction in the upper air passages would occur with more frequency and be recognized. Likewise, the paucity of such reports is impressive. Bretonneau[29] in 1826 advocated and practiced tracheotomy for laryngeal obstruction in membranous croup which he regarded in most cases as diphtheria. Neither he nor his pupil Trousseau[30a] discussed the complication in their earlier manuscripts; however, in 1861 after extensive experience with the operation, Trousseau[30b] described the occurrence of emphysema of the neck and the resulting respiratory embarrassment when it invaded the chest. He emphasized the complication as being promoted by the rapid (stab) method of tracheotomy which he deprecated. In his opinion the more deliberate procedure was preferable, since with it there was less danger of the accident's happening.

Various reasons may be postulated for the apparent failure of these able clinicians to observe the complication which presumably arose occasionally. Many of the patients were moribund when the operation was performed, and died of their disease immediately, or too soon for emphysema to develop. Hemorrhage and failure to relieve asphyxia accounted for most of the operative deaths. Some cases may have been obscured by infection and by the so-called pneumonia, which was often recorded. Certainly a major cause was the inadequate delineation of the diagnostic features of air in the mediastinum, and the fact that the Roentgen ray had not yet been discovered.

Mediastinal Emphysema Complicating Tracheotomy.

The publication of Champney's article was soon succeeded by reports of other cases of post-tracheotomy mediastinal emphysema. Ewart and Roderick[31] in 1899 recorded the case of a five-year-old girl, ill with diphtheria, who died three days after tracheotomy. Autopsy revealed mediastinal and subcutaneous emphysema, which the authors attributed to weakening and bursting of the walls of the pulmonary alveoli from their distension by coughing. They distinguished the two routes by which air may enter the mediastinum in tracheotomy cases, and designated them respectively the extrinsic or inspiratory, by way of the recently divided fascial planes; and the intrinsic or expiratory type caused by distension and rupture of the lung alveoli under stress of bronchiolar obstruction. The latter method they considered to be less common but more serious on account of its supposed uncontrollability.

Leiner[32] in 1903 reported nine cases of mediastinal emphysema following tracheotomy in children. He believed that the air entered the neck wound and extended downward. He gave as reasons for this, the rapid appearance of mediastinal emphysema after the operation, the normal findings in the chest before operation, and the absence at autopsy of interstitial or subpleural emphysema in the lungs of the four patients who died. In 1939 Graebner[33] reported five children tracheotomized for acute laryngeal obstruction, in one of whom the mediastinal emphysema preceded the operation. In the same year, Michels[34] presented six instances of children with either mediastinal emphysema or pneumothorax developing after tracheotomy. Both Graebner and Michels attributed the complication to Macklin's theory. Neffson[35] took the contrary view after a careful discussion of the possible mechanisms in a report published in 1943. This was after an experience with 17 cases of pneumothorax and 30 of mediastinal emphysema in 126 tracheotomies in a 10-year period for the relief of acute non-diphtheritic obstructive infections of the larynx, trachea, and bronchi. Similar conclusions, namely that air is sucked by negative intrathoracic pressure through the tracheotomy wound along the deep cervical fascial planes into the mediastinum, were evinced in two other contemporary series of cases.

Goldberg and associates[36] reported 18 tracheotomies for croup with 12 mediastinal emphysemas and eight pneumothoraces. Their observations were complemented by experimental work on human cadavers. Forbes and associates[37] studied 120 children tracheotomized for acute laryngotracheobronchitis with an occurrence of postoperative mediastinal emphysema in 25 per cent and pneumothorax in 10 per cent of the patients. They also experimented with dogs and confirmed their beliefs.

During this same period Macklin was conducting his animal experiments and proving to his satisfaction the validity of his intrinsic theory of the mechanism by which air gains access to the mediastinum. His exhaustive treatise[19c] of 1944 detailed the applicability of his theory in explaining the development of mediastinal emphysema in many diverse circumstances unaccountable in any other way. A compelling example pertinent to the subject under discussion is the appearance of mediastinal emphysema before tracheotomy or without tracheotomy in occasional cases of acute laryngeal obstruction. Macklin argued that pulmonary interstitial emphysema was already present from alveolar distension and rupture caused by the respiratory obstruction, and that mediastinal emphysema and possibly pneumothorax resulted from its extension, thereby making the tracheotomy a co-incidental and not a causal element.

The route by which air reaches the mediastinum in the presence of any open neck wound when there is also obstruction to the airway continues to be a subject of much debate. The arguments for both theories are fully discussed in excellent reviews published in the decade from 1940 to 1950 by Goldberg,[36] Neffson,[35,38] Forbes[37] and Macklin.[19c] The truth probably is that both mechanisms operate in different cases and both may be involved together in certain individual instances.

For sake of completeness a third possible route to the mediastinum is mentioned, namely, by trauma to the apical pleura in performing a low tracheotomy. This was suggested by Wiethe[39] but is probably rare, as the injury has seldom been reported. As demonstrated by Macklin[19c] the resultant pneumothorax could only with great difficulty, if at all, rupture

medially into the mediastinum to produce a pneumonomediastinum.

The incidence of mediastinal emphysema complicating tracheotomy is influenced directly and definitely by several well-recognized factors:

1. The Urgency of the Operation as Measured by the Severity of the Dyspnea and Other Obstructive Signs. The powerful inspiratory efforts greatly diminish the intrathoracic pressure and favor the indrawing of air through the open incision in the neck. If an interstitial emphysema has already started in the connective tissue of the lung it may continue to progress even though the obstruction is relieved. Once its need becomes apparent, tracheotomy should be performed as early as possible and not as a last desperate expedient.

2. The Technique Employed and the Difficulty Experienced in Performing the Operation. Champneys[20a] considered the dangerous period to be the interval between division of the deep fascia and the introduction of the tube. Undue delay in this period increases the danger. Widespread dissection in the neck tissues and lifting the pretracheal fascia should be avoided. The incision should not be lower than necessary to avoid entering the superior mediastinum. Suturing of the skin close to the cannula was considered dangerous by Jackson[40] but was discounted by Neffson.[38]

The provision of an adequate airway by means of an indwelling bronchoscope, or Mosher life-saving tube, during the tracheotomy procedure has been universally acclaimed as a measure to minimize the likelihood of developing mediastinal emphysema. Originally advocated by Michels[34] the maneuver has amply demonstrated its value in many subsequent reports. In his carefully appraised series Forbes[37] found the incidence of mediastinal emphysema approximately 70 per cent greater for the non-bronchoscoped group, and pneumothorax eight times greater. The frequency of both complications rose more markedly still in the non-bronchoscoped group with respect to the degree of respiratory distress present at the time of the tracheotomy. Everett[41] in discussing the management of 98 tracheotomies in 1175 cases of laryngotracheobronchitis af-

firmed "the presence of an airway—or a small bronchoscope in the trachea during tracheotomy makes the operation an unhurried, tranquil procedure. It is probably the greatest single factor in lowering the mortality rate of tracheotomy *per se*, and in avoiding mediastinal emphysema and pneumothorax". Yet a few notes of warning concerning the procedure have been recorded. MacCready[42] felt that the additional instrumentation sometimes increased the respiratory efforts with its potential danger of inciting the complication. Wiley and Sugarbaker[43] found introduction of the tube impractical in the presence of many obstructive lesions. Holinger[44] drew attention to the risk of too low a tracheotomy while the head is hyperextended with a bronchoscope in place. When the bronchoscope is withdrawn and the head returns to its normal position the tracheal incision may be within the thorax. Subsequent coughing might then force air into the mediastinum.

3. The Adequacy of the Postoperative Supervision. This revolves mainly on the problem of maintaining constantly a clear airway. Repeated aspiration must be employed, if necessary, to eliminate obstruction in the lower passages. The correct placement of the tube within the lumen of the trachea at all times is of paramount importance. The necessity of close watching for a misplaced or blocked tube was emphasized by Reading[45] "that a patient is wearing a tracheotomy tube does not necessarily imply that he is able to breathe through it, nor does the fact that some air can be felt percolating from the wound in the region of the tube mean that he is breathing through the tube".

4. The Age of the Patient. Children develop the complication much more readily than older individuals. Many explanations have been advanced for this. Children tend to experience more acute degrees of respiratory obstruction than adults. They also present more technical problems to the surgeon. The operative field is smaller, with consequently a relatively longer incision. The greater looseness of the areolar tissue in the region facilitates separation of fascial layers and sometimes even allows the tube to be pushed down in front of the trachea in a struggling uncooperative child.

5. The Clinical Condition Necessitating Tracheotomy. Acute

laryngotracheobronchitis deserves special consideration because this disease accounts for the great majority of tracheotomies, especially in children; and the incidence of a complicating mediastinal emphysema is much higher when the operation is required in its presence than in any other circumstance. Two reasons are given for this: *First*, the disease involves the whole respiratory tract combining an acute upper respiratory obstruction in the larynx with impediment in the lower passages by accumulation of thick secretions; dyspnea may persist from the latter after the former is relieved by the tracheotomy. *Second*, the extreme dyspnea and diffuse bronchopulmonary pathological changes tend to promote the production of pulmonary interstitial emphysema, according to the theory of Macklin. The double hazard of upper and lower respiratory obstruction inherent in acute laryngotracheobronchitis was discerned by Neffson and Wishik[46] as early as 1934, and is now generally acknowledged; in fact, the occasional failure of tracheotomy to afford satisfactory relief of dyspnea and the relatively high incidence of supervening emphysema and pneumothorax in this rather ill-defined disease-complex, has resulted in differing opinions as to the advisability of the operation in some cases. Although in favor of the operation when performed, if possible, as an elective procedure, Briggs and Heseltine[47] recently examined divergent points of view regarding this question.

Series of tracheotomies in patients with acute laryngotracheobronchitis as reported by Goldberg,[36] Neffson,[35] and Forbes,[37] with their high incidence of mediastinal emphysema, are in marked contrast to series where the operation was performed for other less complex obstructive conditions. Figi[48] in reporting 206 consecutive tracheotomies at the Mayo Clinic did not mention the occurrence of either mediastinal emphysema or pneumothorax. Only two of these patients had laryngotracheitis. Davis and associates,[49] in 155 operations, observed mediastinal emphysema as a complication in eight patients and pneumothorax in eight patients among 22 of the total having acute laryngotracheobronchitis. They expressed surprise at the infreqeuncy of the complications in the series as a whole, a fact they attributed to lack of routine postoperative chest roentgenograms. Fitzhugh (1941)[50a] in 100 trache-

otomies which included 10 for acute laryngotracheobronchitis, reported two with pneumothorax, one of which also had recognized mediastinal emphysema. In a later series (1954)[50b] of 150 cases which apparently included none of acute laryngotracheobronchitis, there were three pneumothoraces. Subcutaneous emphysema and mediastinal emphysema were mentioned as often present but of a degree to cause no concern. Evans and Smalldon,[51] in New York Hospital, analyzed 50 cases of mediastinal emphysema from various causes, and found 10 occurred in tracheotomized patients, of whom five had acute laryngotracheobronchitis, and all of these were in patients three years of age or younger.

In recent years tracheotomy has been used successfully in an increasing number of entirely different conditions associated with obstruction by secretions in the lower respiratory tract. The purpose is to facilitate removal of obstructive secretions from the tracheo-bronchial tree and thereby prevent pulmonary complications and anoxia. The idea gained prominence through Galloway's work[52] on bulbar poliomyelitis in 1943, and Priest's subsequent studies[53] of the same disease. The new concept enormously increases the indications for the operation. These now include neurological disorders, severe debility, comatose states, botulism, drug poisoning, tetanus, severe head and chest injuries, and lengthy head and neck operations. Recent studies of the basic physiopathology involved, and reviews of these newer indications, with author references, have been made by Von Leden,[54] Fitzhugh and Morgan[50b] and Taylor.[55] Careful perusal of the original articles of the numerous authors cited revealed the enlightening fact that when tracheotomy was performed more or less as a prophylactic measure and not in the presence of acute high obstruction, the probability of mediastinal emphysema was rarely mentioned as a complication, and then usually as only an inconsequential possibility.

During the 12-year period from 1943 to 1954, inclusive, there were 232 tracheotomies performed on 225 patients in St. Luke's and Roosevelt Hospitals in New York City. The accompanying table classifies the indications for the operation according to the degree of respiratory difficulty. Not all the

acute conditions represent emergency procedures with little regard to sterile technique, but all the patients in this category had dyspnea and other signs of definite obstruction at the time of operation. This study is in no sense a statistical one dealing with averages or medians, but rather one concerned primarily with the unexpected and the unpredictable. Yet it is deserving of notice that all of the eight instances of mediastinal emphysema occurred in the urgent group, and in three of these the patient had acute laryngotracheobronchitis.

MEDIASTINAL EMPHYSEMA COMPLICATING TRACHEOTOMY.

	Adults	Children under 12 years of age	Total
Acute laryngotracheobronchitis	5 (1 M.E.)	14 (2 M.E.)	19
Other acute conditions	53 (2 M.E.)	16 (2 M.E.)	69
Malignant neoplasms of larynx			
Acute conditions	21 (1 M.E.)		21
Elective operations	75		75
Other elective operations	42	3	45
	199	33	232

In this series the number of patients operated upon for acute laryngotracheobronchitis seems disproportionately small. They reflected the total number of admissions for this disease, and are possibly explained by the fact that many of them are confused with diphtheria before admission and are taken directly to hospitals for contagious diseases. Among the elective operations there was none for poliomyelitis, as these cases, during the period reviewed, were being handled in other institutions equipped with special isolation facilities.

Physiopathology.

Mediastinal emphysema may remain confined within the mediastinum, or it may escape into the deep and subcutaneous tissues of the neck, into one or both pleural cavities, or through the diaphragmatic openings into the retroperitoneal space. The amount of air invading the mediastinum determines the

pressure that is exerted. Hamman[18c] emphasized that when sufficient pressure develops, air always escapes from the mediastinum. Escape into the neck and contiguous subcutaneous regions is easily detected and is usually considered evidence of mediastinal emphysema. This inference is on sound premises when emphysema of the mediastinum is the sequel of an initial pulmonary interstitial emphysema. In such cases, as for example, spontaneous mediastinal emphysema and that occurring during expiratory straining against a closed glottis, there is justification in terming the presence of subcutaneous emphysema in the neck as pathognomonic of mediastinal emphysema. The finding is not necessarily diagnostic in wounds of the chest communicating with the lung, or where air enters the fascial planes from the buccal or nasal cavities; likewise, air may leak around a tracheotomy opening or through a fracture site in the larynx and cause subcutaneous emphysema in the neck without the inevitable co-existence of mediastinal emphysema. Notwithstanding, in these latter instances one might profitably heed the admonition

"Look to it well and say you are well warn'd."
—Shakespeare: Henry VI, Part 1, Act II, Sc. 4, l. 103.

After subcutaneous emphysema the next most frequently diagnosed sequel of mediastinal emphysema is pneumothorax. This represents escape of air under pressure through the thin pleural surfaces of the mediastinum into the pleural cavity. This actuality was clearly demonstrated experimentally by Joannides and Tsoulos[56] in dogs, and verified by Keis,[57] and Barrie,[55] and Goldberg[36] in human cadavers. In living patients the critical mediastinal pressure necessary to rupture into the pleural cavity remains to be determined. Goldberg's experiments[36] seemed to indicate that it must be above 20 mm. of mercury. Keiss and Barrie both believed that sufficiently high pressure could be attained to accomplish this in the presence of dyspnea from respiratory obstruction, even though an open neck wound was present. They found no reason to question the belief that air may be sucked through an open wound into the mediastinum and thence enter the pleural cavities. Seidl,[59] on the other hand, from his experiments on dogs and clinical observations on thyroidectomy patients, could not conceive of

sufficient pressure being built up in the mediastinum by this method to cause its rupture into the pleural cavity. If air entered the mediastinum by this route, which he conceded as possible, he claimed that it should be absorbed or escape through the original neck wound or through any drainage opening. Seed[60] concurred, and used the findings to further his argument in favor of Macklin's theory to explain mediastinal emphysema and pneumothorax in thyroidectomy cases.

The importance of these rival theories is emphasized anew in the presence of a complicating tension pneumothorax. If the original source of air were an ipsilateral pulmonary interstitial emphysema from ruptured alveoli, further leakage might be lessened or arrested with compression of the lung by the pneumothorax. This action would be non-operative if the air were entering the mediastinum through the fascial planes of the neck. The prognostic significance of the primary route of entry of air is obvious.

One more aspect of the interrelationship of mediastinal emphysema and pneumothorax should be noticed. The rupture of a subpleural emphysematous bleb on the surface of the lung is often offered as an explanation for spontaneous pneumothorax. A valvular mechanism is invoked to explain positive pressure in the pleural cavity. It seems most improbable for mediastinal or subcutaneous emphysema to result from pneumothorax produced in this way. Much higher pressures are required to rupture the normal visceral pleura than the thin mediastinal pleura. Air passes rather easily from the mediastinum into the pleural cavity, but almost never in the reverse direction, as demonstrated by experimental and clinical observations.[19c,18b] If pulmonary interstitial emphysema is present, it is theoretically possible for air to proceed peripherally toward the surface of the lung,[61] but Macklin[19b] proved that pressure gradients took most of it along the blood vessel sheaths toward the hilus. If air entered by way of the neck the mediastinal emphysema must perforce have preceded the pneumothorax.

The third escape route for air from the mediastinum is through the diaphragm about the aorta and esophagus to the

retroperitoneal tissues. This pathway is utilized less frequently than the superior aperture into the neck. Sweet[62] explained this by the fact that air in body spaces tends to rise in contradistinction to liquids which gravitate downward. He speculated that this physical law applied in mediastinal emphysema when the patient was in the usual semi-recumbent position, and that it accounted for the more common appearance of emphysema in the subcutaneous tissues of the neck.

A small amount of air in the mediastinum may produce only trivial symptoms, or none at all; whereas large amounts may cause grave disturbances and may even lead to death. Jehn and Nissen[63] proved by animal experiments that no recognizable symptoms appeared until the normally negative intramediastinal pressure reached about zero. If more air was introduced signs of cardiac tamponade, falling blood pressure with dyspnea and cyanosis soon supervened. These changes progressed rapidly depending on the amount of pressure and the rapidity of its increase. The effects were the result of increased mediastinal pressure causing mechanical interference to the circulation by compression of the large veins entering both sides of the heart. This action was demonstrated experimentally in rabbits by Ballon and Francis[64] and confirmed clinically by Jessup.[65] That the pernicious consequences of increased mediastinal pressure as noted by these investigators was due to the rapidity and the uncontrolled extent of its formation is illustrated by comparison with the benign reactions observed during the induction of orderly diagnostic artificial pneumomediastinum.

The diagnostic injection of 40 to 150 cc. of air into the mediastinum of each of 40 children by Castellanos and Pereiras[66] caused no ill effects. In adults, Bariéty and associates[67] routinely injected 400 to 700 cc. of air in 20 minutes, creating a final pressure of 8 to 15 cm. of water with only insignificant symptoms. They attributed this remarkable tolerance to avoidance of sudden and massive distension of the mediastinal cellular tissue with resultant flooding of the escape avenues. The availability and spontaneous use of these escape channels thus constitutes a powerful factor in regulating abnormal intramediastinal pressures.

Torrey and Grosh[68] noted the dramatic relief of symptoms and prompt improvement in the patient's condition as high intrathoracic pressure was relieved by the appearance of subcutaneous emphysema. Conversely, it has been often commented that when there are alarming signs of increased mediastinal pressure there is usually little subcutaneous emphysema. A reciprocal relationship between mediastinal pressure and subcutaneous emphysema was noted experimentally by Ballon and Francis.[64] The release of air under pressure from the mediastinum into the pleural cavity does not offer such a safe escape. The capacity of the pleural cavity is limited, and dyspnea and cyanosis are augmented rather than relieved if tension pneumothorax supervenes. If the condition is bilateral, life itself is in grave peril. In his monograph on acute laryngotracheobronchitis, Neffson[38] estimated the mortality to be less than 20 per cent in unilateral pneumothorax, but about 90 per cent in bilateral cases.

Much has been learned about the anatomy of the region since the time of Ambroise Paré and the succeeding Seventeenth Century. The mediastinum is no longer believed to be the firm rigid structure described by Crooke[69] in 1616, whose action is to support and steady the heart and to separate it from the lungs, and the lungs from each other, so that all may function independently. Increased knowledge of the physiology of the mediastinum was made possible by the introduction of differential pressure anesthesia in 1904 by Sauerbruch.[70] A means was thus provided for maintaining respiration when the chest had been laid open, and one or both lungs were exposed to the atmospheric pressure, thereby permitting operations to be performed on thoracic viscera. Surgeons could then verify clinically Graham's modern concept[71] of a yielding and mobile mediastinum and contents; yet much is still unknown regarding the physio-pathology of the mediastinum. Bariéty[67] has epitomized it well by stating that one does not deal here with a well-defined closed space to which accurate physical laws may be applied, but rather with a passage open upwards and downwards, whose size, capacity, structure, suppleness and permeability are variable from one subject to another. These variables must be taken into account when evaluating an individual case of mediastinal emphysema.

Symptoms and Physical Signs.

Once one is familiar with the syndrome of mediastinal emphysema and its sequelae its identity should not be difficult. The diagnosis depends primarily upon the surgeon's awareness of the possibility. Symptoms usually appear during the operation or within a few hours of its termination. The clinical signs have been described[72,73,74] more comprehensively in discussions concerning spontaneous emphysema of the mediastinum in which the picture is clearly defined and the symptoms are not mingled with those of an operation or an antecedent medical condition. Schwartz, McIlroy and Warren[75] observed the essential similarity in the clinical picture of all cases of mediastinal emphysema, however caused, but noted that any fatal cases have invariably occurred in the secondary type.

Pain in the chest is the most constant symptom and is characteristically precordial or substernal. It may be severe with anginal type of radiation simulating organic heart disease.[76] This has been noted particularly in spontaneous cases but according to Scott[77] it is a feature in any serious increase in mediastinal pressure from whatever cause. Pain of this sort and severity necessitates differentiation from the two diseases most commonly confused with the entity: acute pericarditis, and acute coronary occlusion with myocardial infarction. In mediastinal emphysema the pain typically alters with changes of position of the body, varies with respiration, and may be aggravated by swallowing or turning the head. Surgeons are aware of the significance of this kind of pain when it occurs in chest injuries. In these traumatic cases Gatellier[78] emphasized the importance of searching for a developing mediastinal emphysema at the onset of diaphragmatic pain, especially when associated with unexplained dyspnea.

Dyspnea in some degree is nearly always present, and as indicated by Jessup[65] is directly related to the extent of embarrassment of vital structures in the mediastinum by trapped air. When very severe and accompanied by cyanosis, pneumothorax should be suspected. That mediastinal emphysema alone may be responsible, however, must be borne in mind. The experience of Torrey and Grosh[68] showed that severe dyspnea and cyanosis materially lessened when air escaped

into the subcutaneous tissues of the neck. Gumbiner and Cutler[79] established relief of these symptoms in newborn children by removing air from the mediastinum by a needle.

No constitutional signs usually accompany the syndrome except in severe cases. Draper[73] said that this fact provided negative evidence of inestimable value in distinguishing mediastinal emphysema from more serious cardio-vascular disorders. There is no associated fever, leucocytosis, or increased sedimentation rate. Until the condition is severe and, therefore, otherwise obvious, there is no change in the pulse rate or blood pressure. Electrocardiographic changes may occur[72,76] but they are inconstant, transient, and not characteristic.

The most distinctive physical sign of mediastinal emphysema is mediastinal crepitation (Hamman's sign). This consists of crackling, bubbling or crunching sounds heard over the heart with each contraction. These may be heard only during systole, but sometimes they are present throughout the whole cardiac cycle with accentuation during systole. They may vary greatly during the episode and with change of position or phase of respiration. The sounds are typical and do not occur with any other condition. When present the sign is pathognomonic. Its absence, however, does not rule out emphysema of the mediastinum. The air must have migrated to the anterior mediastinum to be heard. In Macklin's experiments[19b] air tended to accumulate first in the middle and posterior mediastinal spaces. The sign usually persists for six to eight days, but may last only a few hours and thereby be missed. It is less constantly observed in children than in adults. Goldberg[36] found Hamman's sign in only six of 31 children with proven mediastinal emphysema.

Diminished or obliterated cardiac dullness, sometimes replaced by hyperresonance, is less frequently reported, but its presence has diagnostic value.

Subcutaneous emphysema has long been acknowledged as a common and reliable sign of mediastinal emphysema. It is easily recognized and is often the first intimation that air is contained within the mediastinum. It usually appears first in the neck, thence spreading over the face and thorax, and may

finally become generalized over most of the body. In the absence of an open chest injury or a penetrating wound of the upper air passages, it must be considered presumptive evidence of mediastinal emphysema. Subcutaneous emphysema is the palpable sign of a mechanism by which intramediastinal pressure is relieved. Its extent, therefore, is no accurate gauge of current conditions in the mediastinum. The important matter is the quantity of air still retained in the mediastinum.

Pneumothorax, as a sequel to mediastinal emphysema, must by definition, follow it; but when pneumothorax does develop, it is discovered simultaneously in the majority of cases. Occurring with operations in the neck, mediastinal emphysema and pneumothorax probably have an identical origin, and because of their frequent conjunction they are usually considered as parts of the same entity. The dramatic signs of pneumothorax, often sudden and accompanied by shock, overshadow the lesser ones of a preceding mediastinal emphysema. During an operation or in the post-operative period while the patient may be still under the influence of anesthesia, the onset of pneumothorax may provide the first and only evidence of air under pressure in the mediastinum. The gravity implicit in the diagnosis should not be underestimated.

Roentgenographic demonstration of air in the mediastinum is pathognomonic of emphysema. Anterior-posterior views show widening of the mediastinal shadow with irregular dark streaks parallel to the cardiac border on either side. Small amounts of air, unless localized, may not be visualized on this view. Aisner and Franco[74] directed attention to the error of eliminating mediastinal emphysema on the basis of a single film taken in the postero-anterior projection and advised serial films if necessary. Like Gumbiner and Cutler[79] they stressed the value of lateral projections in revealing air behind or in front of the heart. Evans and Smalldon[51] considered lateral and oblique views essential before one could rule out mediastinal emphysema. Seed[60] was of the same opinion but pointed out the difficulty of obtaining a picture in a dyspneic bed-patient by means of a portable X-ray machine. Schinz, Boensch, Friedl and Uehlinger,[80] in their recent textbook of Roentgen diagnosis, recommended fluoroscopy of the mediastinum in the

oblique position. The awkwardness of using this technique with an ill postoperative patient is a disadvantage.

The importance of routine Roentgenograms of the chest to detect mediastinal emphysema and pneumothorax after operations on the neck cannot be over-emphasized. Some of the patients with these complications present few or no symptoms, and the condition would otherwise remain unrecognized. Davis[49] believed numbers of cases are missed in this manner. Forbes[37] advised examination by fluoroscopy or by Roentgenogram within six hours in all tracheotomy cases. In his series the incidence increased from 25 per cent to 41 per cent in the case of mediastinal emphysema and from 10 per cent to 15 per cent in that of pneumothorax when the patients were checked by Roentgen examination.

REPORT OF TRACHEOTOMY CASES.

Case 1: E. P., 1954. Acute Laryngotracheobronchitis. This 10-year-old white boy had fever, cough, and increasing dyspnea for three days. On admission his temperature was 103° F., pulse 140 and respirations 50 and irregular. Dyspnea was severe with cyanosis and chest retraction. Roentgenographic examination of the chest immediately before tracheotomy was negative. Rapid improvement in symptoms occurred after the operation. On the following day marked subcutaneous emphysema was present in the neck and anterior chest wall. Hamman's sign was positive, and a Roentgenogram revealed air in the mediastinum but none in the pleural cavities. The child was given oxygen, steam inhalations, and antibiotics. The air disappeared from the mediastinum in three days and from the subcutaneous tissues in five days. The tracheotomy tube was removed on the sixth day, and the patient was discharged from the hospital as cured on the tenth day.

Case 2: M. W., 1953. Acute Laryngotracheobronchitis. A 21-year-old white woman had acute respiratory symptoms for 36 hours. Her temperature was 103° F., pulse 100, and respirations 22 on admission. Marked inspiratory stridor and extreme dyspnea were present. Preoperative Roentgenogram of the chest was negative. Tracheotomy afforded relief of the dyspnea. Subcutaneous emphysema in the neck and upper part of chest appeared the next day. Positive signs of mediastinal emphysema were verified by Roentgenographic examination. No pneumothorax occurred. The patient was treated with antibiotics. The tube was removed in six days. At that time examination of the chest was negative, but there was still some subcutaneous emphysema in the neck and external chest wall.

Comment: Two cases of mediastinal emphysema following tracheotomy for acute laryngotracheobronchitis are presented: one in a child and one in an adult. Both were of moderate degree and both exhibited subcutaneous emphysema without pneumothorax.

Case 3: A. M., 1943. Acute Laryngotracheobronchitis. A 2-year-old white boy was acutely ill with fever and dyspnea. He had been in the hospital a year previously for a similar but milder attack, and had been treated only medically. This time the child had been ill for one week and on admission was extremely dyspneic and in poor condition. Roentgenograms of the chest showed slight patchy consolidation in both lungs and a small pneumothorax at the right base. No mediastinal emphysema was noted, but a lateral view was not taken. Tracheotomy was performed with a bronchoscope in place in the trachea. Dyspnea was only moderately relieved by the operation and was not improved by tracheal suction. The patient became cyanotic, and died suddenly one-and-a-half hours after the operation. Permission for autopsy was not granted, but the clinical signs on physical examination signified a bilateral tension pneumothorax. No subcutaneous emphysema was present.

Comment: This is a case of acute fulminating laryngotracheobronchitis with evidence of pneumothorax before tracheotomy, suggesting the presence of pulmonary interstitial emphysema according to Macklin's theory. It illustrates the potential seriousness of mediastinal emphysema when the pleural cavity is utilized as the escape route for the aberrent air under pressure.

Case 4: J. L., 1943. Impacted Foreign Body in the Larynx. This 3-year-old white boy claimed he had swallowed a nut which a stranger had given him just before he was brought to the hospital. Distinct wheezing and some dyspnea were present. Breath sounds were diminished on the right side. Roentgengraphic examination did not demonstrate a foreign body but did reveal a slight obstructive emphysema of the entire right lung. Without anesthesia a 5 mm. bronchoscope was readily introduced, and a smooth hard-shelled nut was found obstructing the orifice of the right main bronchus. Difficulty was experienced in grasping the foreign body satisfactorily. During withdrawal it was dislodged from the forceps in the subglottic region. Severe laryngeal spasm immediately ensued, and the patient's airway was completely obstructed. Emergency tracheotomy was performed and normal respirations were resumed. During the operation and before the lumen of the trachea was entered the child made several powerful inspiratory efforts. The foreign body remained so tightly wedged between the cords that it could not be readily extracted. The decision was made to leave it in place above the cannula for a while rather than to administer a general anesthetic to facilitate its removal. The lower air passages and lungs were clear. Several hours later, during the night, slight subcutaneous emphysema in the suprasternal notch was noted, but no other abnormal signs were present. Oxygen was given, although no dyspnea was observed. Sixteen hours after the operation severe dyspnea and cyanosis suddenly supervened. Bilateral tension pneumothorax was diagnosed and both pleural cavities were aspired with needles and syringes, but the patient went into shock and died. Autopsy was performed under water five hours after death. Subcutaneous emphysema was present in the neck and right supraclavicular fossa. The mediastinum contained multiple blebs of air distending its entire connective tissue structure. Both lungs were collapsed by tension pneumothorax. All pleural surfaces were smooth, showing no emphysematous blebs and no signs of injury. The pleurae revealed no adhesions. There was no evidence of trauma to the parietal pleura or to either lung at the apex. The foreign body proved to be a

pistachio nut in its shell measuring 14x8x7 mm. in size. It fell free into the trachea during the post-mortem examination. There was no indication of laceration or other injury of the bronchi or of the trachea below the tracheotomy site. The microscopic examination of the lungs revealed almost complete atelectasis of both lungs. Some alveoli were distended, and a few showed rupture of their walls. Careful search, however, failed to disclose any air in the interstitial tissues of the lungs or in the perivascular or peribronchial sheaths.

Comment: Direct aspiration of air through the open neck wound during the operation was thought to be the cause of the mediastinal emphysema in this case. Why the pressure continued to build up by this route in the face of a subsequently clear airway is not readily explained. Pulmonary interstitial emphysema would satisfactorily reconcile the sequence of events, but no confirmatory evidence could be elicited by post-mortem examination which was conducted under optimum conditions.

Case 5: J. G., 1951. Carcinoma of Larynx with Edema. Hoarseness for nine months and dysphagia for two weeks had not impelled this 63-year-old white laboring man to seek medical advice, but dyspnea for 24 hours did. Acute edema of the larynx superimposed on a large carcinoma necessitated an emergency tracheotomy. During the procedure the patient was severely dyspneic and had extreme retraction of the chest. Several sutures were inserted to close a rather long incision. One hour after the operation subcutaneous emphysema appeared in his neck and chest wall. Increasing dyspnea pointed toward mediastinal involvement. Prompt removal of the sutures was accompanied by escape of air and relief of symptoms. Signs of both mediastinal emphysema and subcutaneous emphysema were gone in six days.

Comment: The evidence in this case suggests that the emphysema was caused by too tight closure of the incision around the tracheotomy tube. Jackson[40] has warned about this danger.

Case 6: J. M., 1953. Acute Edema of Larynx Superimposed on Post-radiation Fibrosis. An emergency tracheotomy was performed on this 46-year-old white man immediately after admission to the hospital. This was made necessary by an acute edema of the larynx with obstruction causing stridor, dyspnea and some cyanosis of a few hours' duration. History obtained later revealed that he had had a tracheotomy performed in another hospital 10 years earlier for carcinoma of the larynx. The operation had been followed by prolonged treatment with radium and the patient had worn the tube for seven years. No symptoms except chronic hoarseness for the last three years were noticed until the day of his present admission.

Tracheotomy was extremely difficult because of the excessive degree of dense fibrosis in the tissues of the region. This obviously was a sequel to the previous operation and subsequent radiation therapy. Considerable bleeding was encountered which further delayed a troublesome dissection. The following day subcutaneous emphysema was evident in the neck and right side of the chest. The patient complained of considerable pain in the substernal region, but had no elevation of temperature or leucocytosis. Roentgenographic examination revealed a pneumo-

thorax at the left base and a possible mediastinal emphysema (no lateral view taken). Symptoms improved with disappearance of signs of abnormal air in 12 days. The patient left the hospital with the tracheotomy tube in place. Direct laryngoscopy, afterward, revealed distortion and partial cicatricial stenosis of the larynx, but no evidence of regrowth of neoplastic tissue.

Comment: Widespread dissection of the neck tissues and delay in entering the trachea in the presence of acute laryngeal obstruction predispose to the development of mediastinal emphysema in tracheotomy cases. When such technique is unavoidable prudence calls for increased vigilance during the postoperative period.

Case 7: R. H., 1954. Revision of Tracheotomy. When two months old, this Negro boy had a herniorrhaphy performed for an incarcerated congenital left inguinal hernia. While reacting from the anesthesia (open inhalation method) he regurgitated and aspirated some gastric contents. Bronchial aspiration by catheter and also through a 3½ mm. bronchoscope failed to relieve the dyspnea and cyanosis, so an emergency tracheotomy was done, using a No. 1 tube. The acute condition was alleviated. Subsequent attempts to decannulate the trachea were unsuccessful because of chronic subglottic edema causing obstruction above the tracheotomy opening. This was attributed to too high placement of the tube in the trachea. One month after the primary operation, the tracheotomy wound was revised and the cannula inserted considerably lower in the airway. The operation was performed under general ether anesthesia administered by mask over the open tracheal wound and under no pressure. No difficulty was encountered with the anesthesia. Immediately after completion of the operation extensive subcutaneous emphysema appeared in the neck and upper part of the chest, and the patient became slightly dyspneic. Roentgenograms showed extensive emphysema of the mediastinum and subcutaneous tissues of the neck and chest wall, with clear lungs and no pneumothorax. Preoperative films had been negative. It required nine days for the air beneath the skin and in the mediastinum to disappear clinically and as shown on Roentgen films.

Comment: The mediastinal emphysema in this case was probably caused by the incision and dissection being carried too low in the neck, thereby opening the fascial planes close to the superior mediastinum. Pulmonary interstitial emphysema associated with the anesthesia cannot be ruled out, but no positive pressure was used and no dyspnea was observed during the operation.

Case 8: J. S., 1954. Resection of Trachea for Stenosis caused by Tracheomalacia. Two months before admission this 60-year-old white woman had had a thyroidectomy performed in another hospital. A large substernal gland diagnosed as Hashimoto's struma had been excised under general anesthesia without tracheal intubation. The immediate postoperative period was stormy with much dyspnea and cyanosis. The dyspnea had persisted in severe degree and was accompanied by loud expiratory wheezing, characteristic of tracheal obstruction.[81] Her voice was good, and vocal cord function was normal. Bronchoscopy revealed a

smooth concentric stenosis of the trachea at the level of the fifth ring. This region dilated during inspiration but during expiration collapsed to a caliber which would barely permit passage of an ordinary bronchoscopic suction tube. Routine Roentgenograms of the chest were negative. Laminographs showed the trachea to be in the midline with a smooth tapering tracheal stenosis opposite the second and third thoracic vertebrae, and, in addition, showed a poorly defined bilateral soft tissue density in the substernal region.

Under local anesthesia the original transverse neck incision was reopened and the trachea exposed. The second and third rings were cut for the insertion of a rubber breathing catheter extending past the site of the stenosis. Much dense fibrous scar tissue made exposure and freeing of the trachea very difficult. The fifth tracheal cartilage had been destroyed and was replaced by a band of fibrous tissue which constricted the lumen to ½ cm. in diameter. This area for a distance of ¾ cm. was resected, and the upper and lower segments of the trachea were approximated with interrupted sutures of No. 32 stainless steel wire. The anastomosis appeared to be air-tight. The tracheal lumen could now accommodate the No. 5 tracheotomy tube, which was put in place through a new opening in the skin above the orginal incision. The primary wound was closed and two small Penrose drains were inserted. Biopsy of the residual thyroid tissue later confirmed the diagnosis of Hashimoto's type of chronic thyroiditis.

Subcutaneous emphysema appeared in the neck on the day following operation. Roentgenographic examination revealed slight emphysema in the mediastinum and in the fascial planes of the neck. This disappeared in five days. Other symptoms were absent. No pneumothorax occurred. The tracheotomy tube was removed in ten days. Subsequent bronchoscopy demonstrated a normal tracheal lumen. The patient has remained well.

Comment: This is an example of mediastinal emphysema occurring after a difficult neck dissection and tracheotomy in the presence of respiratory obstruction. The case might logically be included among those occurring during thyroidectomy. It was considered improbable that air was blown directly through the site of the tracheal resection because the dyspnea had been completely and permanently relieved before the anastomosis was accomplished.

Mediastinal Emphysema Complicating Thyroidectomy.

In 1946 Meek[82] remarked that for mediastinal emphysema to follow surgical procedures on the neck was a logical conclusion if one considered both the relationship of the cervical fascial spaces to the mediastinum and the mechanics of breathing. The first report of mediastinal emphysema complicating a neck operation other than tracheotomy was by Buford[83] in 1918. The procedure was a thyroidectomy and excision of tuberculous nodes of the neck. In 1924 Gold[84] reported four cases following thyroidectomy. In 1934 Keis[87] collected seven cases in the literature; all followed thyroidectomy; all had associated

pneumothorax; all were fatal. In subsequent years there have appeared sporadic reports of other cases, nearly always accompanied with pneumothorax and nearly always extremely serious. The low incidence and extreme gravity of these reported cases has led to the suspicion that mediastinal emphysema complicating neck operations has occurred much more frequently than the literature would indicate and that the complication often might be mild and unrecognized.

Next to tracheotomy, thyroidectomy has accounted for most of the cases of mediastinal emphysema occurring as an operative complication. Operations on the neck, other than thyroidectomy, however, have been reported as the responsible agent. The factors involved are analogous. The case reported by Schmidt[85] in 1931 followed a jugular vein ligation; that by Ackerman and Bricker[86] in 1941 followed a radical dissection of cervical lymph nodes, and one of the two by Herrmann[87] in 1942 followed resection of the upper segment of the esophagus for carcinoma. Additional cases in the literature to 1949 have been listed by Seed.[60] Since that year two significant series of cases have appeared. Bowden and Schweizer[88] in 1950 reported 18 cases of pneumothorax and two of mediastinal emphysema arising during operative procedures about the head and neck in a five-year period at Memorial Hospital, New York. Aiken and Smith,[89] in England in 1953, found either mediastinal emphysema or pneumothorax as a complication in eight patients among 900 block dissections of the neck. An increased incidence of the complication or an increased awareness of its development, or both, must be inferred.

The theory of the mechanism by which mediastinal emphysema develops in thyroidectomy cases, as in tracheotomy cases, has been the subject of much debate. The majority of the earlier investigators favored the theory that air entered the neck wound and was sucked into the mediastinum along the fascial planes by negative pressure. Barrie[58] had three patients who died of mediastinal emphysema and pneumothorax following thyroidectomy. The autopsy findings and his subsequent experiments on cadavers convinced him that air entered by way of the wound. Keis[57] had previously performed similar experiments and reached the same conclusion. Ackerman and

Bricker[56] were strongly of the same opinion. Like Keis they found no post-mortem evidence of pulmonary interstitial emphysema in their patient. Aiken and Smith,[59] after discussing the possible routes of entry, also concluded that air entered by way of the neck, but they restricted the pathway to the carotid sheath. They stressed the sucking sound heard at operation as being indicative of this mechanism. Barrie[58] had also described this sound and thought that it signified air being drawn into the tissue spaces.

The important role of respiratory obstruction in the development of mediastinal emphysema during neck operations was early appreciated. Schmidt,[55] and Jessup[65] both stressed the danger of inspiratory dyspnea from tracheal compression. As long ago as 1924 Gold[54] suggested having a tracheoscope in place during the operation to overcome upper tracheal obstruction. Pressure by a goiter is not the only cause of dyspnea in thyroid operations. Edema, hemorrhage into the wound,[90] excessive mucus, surgical interference with the recurrent laryngeal nerves,[45] laryngeal spasm,[91] tracheal collapse,[45] and intubation difficulties[85] have all been blamed. For significant amounts of air to enter the mediastinum directly along the fascial planes from an open neck wound seems to require the simultaneous existence of some degree of respiratory obstruction.

Inextricably connected with the issue of mediastinal emphysema in thyroid surgery is the element of anesthesia. These operations are customarily performed under general anesthesia with the agent administered sometimes by the inhalation method but commonly through an intratracheal tube and under positive pressure. Coughing and signs of tracheal and bronchial obstruction are familiar incidents during these procedures. The tendency for interstitial emphysema of the lung to develop under these conditions is well established. The onset of emphysema of the mediastinum is a logical sequence. In more than half the cases of mediastinal emphysema during thyroidectomy analyzed by him, Seed[60] noted difficulty in breathing either as a result of removing an intrathoracic goiter or from trouble with the anesthesia. Seidl[59] emphatically attributed mediastinal emphysema in thyroidectomy patients to

obstruction of respiration during the operation. He believed the increased pressure caused alveolar distension and rupture leading to pulmonary interstitial emphysema.

The intrapulmonary pressure developed during intratracheal insufflation anesthesia may be the determining factor. Too high a pressure of gas may be entering the lungs. In his experiments on cats and dogs, Macklin[19a] conclusively demonstrated the effects of high intrabronchial pressure. Air could break through the overdistended alveoli into the interstitial tissue of the lung and thence track along the perivascular channels to the hilus and rupture into the mediastinum. That surgical emphysema can develop in this way from anesthesia alone was forcefully illustrated in Bradshaw's patient.[92] Symptoms of the condition, including a small pneumothorax, appeared after intubation and before any operation was attempted. As Macklin[19c] said, some cases of pulmonary interstitial emphysema and pneumomediastinum attributed to the surgical procedure in reality arise during the administration of the anesthesia. Marcotte, Phillips, Adams and Livingstone[93] confirmed that intrabronchial pressure of 24 mm. Hg. could produce mediastinal emphysema in dogs. Such a rise might be caused by coughing or by any obstruction to the outflow of gas. Important to the subject under discussion is Marcotte's conclusion that intrabronchial pressure depended chiefly upon the patency of the outlet rather than upon the pressure at which the gases were forced into the lungs, and the observation that once emphysema had developed, it might be increased at a pressure lower than that necessary to initiate it. Spence[94] considered the administration of an anesthesic at a pressure of 20 mm. Hg. as unsafe. He advised a pressure of 10 mm. Hg. or less when using an intubation technique.

Injury to the mucosa of the upper air passages is often alluded to as a possible route of entry for air into the mediastinum. Such an injury might be a laceration occurring during the insertion of an intratracheal tube. Air then could be blown directly into the mediastinum and rapidly distend it. Aiken and Smith[89] believed that the accident occasionally happened and they cited examples from the literature. A feature was the rapid development of subcutaneous emphysema after induction

of anesthesia. Most writers on the subject have dismissed this course as unlikely and have not noted such injuries.[60,86] Even if a mucosal laceration were present, Macklin[19c] advised careful search for interstitial emphysema of the lungs as the more probable source of mediastinal air. Despite the potential hazards of intratracheal anesthesia, Meek[52] thought the free airway which the tube afforded was good prophylaxis against the production of mediastinal emphysema.

The fundamental problem in thyroidectomy is essentially the same as that in tracheotomy. An open neck wound exists in the presence of an inadequate airway. Many tracheotomy patients are children with their greater susceptibility for developing mediastinal emphysema and in whom the degree of preoperative dyspnea is often alarming. Thyroidectomy, on the contrary, is performed chiefly in adults, but the operation is more prolonged, and it is subject to many intercurrent factors tending to promote respiratory obstruction during the procedure. The occurrence of mediastinal emphysema during thyroidectomy and comparable neck operations has generated discussion paralleling that occasioned by the same complication in tracheotomy. Conflicting theories of the mechanism of production of mediastinal emphysema during these operations have been expounded in several recent articles.

Barrie,[58] Keis,[57] Ackerman and Bricker,[86] and Aiken and Smith[59] all concluded that the air entered the mediastinal tissues by way of the neck wound. They found no evidence of injury to the mucosa of the upper respiratory tract that would permit direct entrance of air into the mediastinum. Neither did they find any injury to the apical pleura. Pulmonary interstitial emphysema was eliminated by consistently negative findings at autopsy.

Seed[60] ably discussed the evidence in favor of Macklin's theory. He quoted the observations and experimental findings of Seidl[59] who was also convinced that this mechanism was responsible for the complication. Although aspiration of air through the open wound was the most frequent explanation given as the cause of mediastinal emphysema and pneumothorax following thyroidectomy, Seed found that it could not satisfactorily explain all cases; moreover, the failure to dem-

onstrate pulmonary interstitial emphysema and mediastinal emphysema at autopsy in all patients believed to have had these conditions had been reconciled by Macklin[19c]. He had emphasized that the diagnosis might easily be missed unless a special underwater technique was employed. He had stressed, further, that if the examination were delayed a number of hours, the air in the interstitial tissues of the lung and in the mediastinum would diffuse and not be discovered. Concerning Macklin's theory, Seed asserted "this theory will satisfactorily explain the history and findings of all the reported cases of mediastinal emphysema and pneumothorax following thyroidectomy. No other one will."

Bowden and Schweizer[68] presented an excellent summary of the anatomical considerations relating to these complications in neck surgery. By opening the middle layer of the deep cervical fascia which envelops the thyroid gland, a free passageway into the mediastinum is provided. Between the middle and deep layers of the deep fascia in the midline space containing the trachea and esophagus there is a direct passage into the mediastinum. After analyzing their 20 cases they thought that in two patients mediastinal emphysema and and pneumothorax followed rupture of the marginal alveoli causing pulmonary interstitial emphysema; but in the majority of cases they were convinced that the air entered the superior mediastinum through the operative site during exaggerated respiratory effort.

The diagnosis of mediastinal emphysema occurring alone can rarely, if ever, be made during the course of a neck operation. The classical signs and symptoms cannot be elicited, or are masked by the anesthesia. Even in the postoperative period the isolated complication is seldom reported as a diagnosis. The condition is usually identified by the appearance of air in one of the escape channels. This circumstance brings conviction that many cases of mediastinal emphysema of mild degree have escaped recognition. Subcutaneous emphysema in the neck may occur during or immediately following the operation and is presumptive evidence of mediastinal emphysema. Tension pneumothorax primarily creates the symptoms which direct attention to the lungs and mediastinum. A characteris-

tic sign[60,88] of evolving pneumothorax is some variation in the patient's normal respiratory pattern during the operation. In the presence of positive tension within the pleural cavity there is typically a relatively long expiratory phase alternating with a very short inspiratory phase. Severe dyspnea and cyanosis may supervene. At the onset of such signs the surgeon should suspect the presence of mediastinal emphysema and pneumothorax.

REPORT OF THYROIDECTOMY CASES.

During the 12 year period from 1943 to 1954, inclusive, 1651 thyroidectomies were performed in St. Luke's and Roosevelt Hospitals in New York City. Mediastinal emphysema occurred as an operative complication in three of these cases.

Case 9: W. W. Operation January 14, 1947. Subtotal thyroidectomy was performed on this 56-year-old white man for removal of a diffuse toxic goiter. Preoperative medication of propylthiouracil for six weeks and Lugol's solution for one week had lowered his basal metabolic rate from plus 53 per cent to plus 24 per cent. The operation was done under general inhalation anesthesia using an endotracheal tube. Operating time was 90 minutes. No difficulty was experienced with the anesthesia or the operation. A small Penrose drain was inserted. Three hours after completion of the operation, the patient had severe pain in the chest, mostly in the substernal region and on the right side. Dyspnea, cyanosis and tachycardia rapidly supervened. Physical examination revealed loud crepitations in the precordial region during systole (Hamman's sign) and signs of tension pneumothorax on the right side with extreme mediastinal shift to the left. Subcutaneous emphysema was present in the neck, anterior chest wall, right axilla and right posterior chest wall. Roentgenographic examination confirmed the diagnosis of mediastinal emphysema and right pneumothorax with total collapse of the lung. Immediate underwater drainage of the right pleural cavity was instituted and was continued for two days. The patient improved rapidly. Hamman's sign persisted for three days. On the sixth postoperative day a Roentgenogram showed almost complete expansion of the right lung. Slight subcutaneous emphysema was still present seven days after the operation. On the tenth day the patient was out of bed and without symptoms.

Comment: This case is somewhat unusual in that no difficulty was encountered with the operation or with the anesthesia. It illustrates the fact that the complication is unpredictable and that its occurrence cannot always be anticipated. The efficacy of prompt and adequate decompression for the relief of tension pneumothorax is demonstrated.

Case 10: M. H. Operation October 20, 1950. This 41-year-old white man, a mechanic, had a subtotal thyroidectomy for a diffuse toxic goiter that had caused symptoms for one-and-a-half years. Administration of propyl-

thiouracil for 14 months had lowered his basal metabolic rate from plus 64 per cent to plus 4 per cent. Lugol's solution was given for one week before the operation. A very large goiter required two hours for its removal. Intravenous pentothal and closed gas-oxygen-ether inhalation anesthesia was employed without tracheal intubation. During the second half of the operation irregular respirations for 30 minutes were noted and attributed to light anesthesia. The skin incision was closed without drainage.

A few hours after the operation the patient had slight precordial pain, but no other symptoms except traces of subcutaneous emphysema in the neck. The pain persisted until the third postoperative day, when it became severe and was accompanied by moderate dyspnea and increased pulse rate (90 per minute). No cyanosis was observed. The pain shifted to the substernal region and did not radiate. It varied with respiration and with body position, being worse on deep inspiration and being relieved by a sitting posture. It was made worse by swallowing. These symptoms lasted four more days and then disappeared. The patient was well and able to leave the hospital on the eighth postoperative day. During the period of his acute pain, his temperature and blood pressure remained normal. Blood count and sedimentation rate were normal. Physical examination of the heart and lungs revealed no abnormalities. No relief was obtained by the administration of nitroglycerine. An electrocardiogram obtained on the fourth postoperative day showed R. T. segment changes suggestive of pericardial disease. A subsequent tracing three days later showed changes consistent with subsiding pericardial involvement. On the basis of these findings the medical consultant ruled out coronary or myocardial disease. A provisional diagnosis of pleuropericarditis secondary to mediastinal emphysema or to a low-grade mediastinitis was made. In the absence of any constitutional signs of inflammation and with the prompt subsidence of symptoms the presence of mediastinal emphysema was considered more likely. Unfortunately, although Roentgenographic examination was suggested, no films were obtained.

Comment: The diagnosis of mediastinal emphysema was not proved absolutely in this case but the clinical findings, both positive and negative, cannot be explained by any other condition. The clinical picture is reminiscent of that described[72,75] in some cases of spontaneous mediastinal emphysema simulating cardiac disease.

Case 11: I. B. Operation December 15, 1952. Eighteen years previously, this 39-year-old white woman had had a partial thyroidectomy for exophthalmic goiter. She now had a recurrent non-toxic nodular goiter with suspected malignant changes. Total removal of a large hard right lobe was accomplished by operation under general inhalation anesthesia with an endotracheal tube in place. Intubation was difficult. The operation required two-and-one-quarter hours and during the latter half the patient had a severe spell of coughing lasting 10 minutes. A rubber tissue drain was left in place after the skin incision was closed. Precordial pain began a few hours after completion of the operation. It was moderately severe, did not radiate, and was increased by movement and by coughing and swallowing. Crepitations synchronous with the heart beat (Hamman's sign) were audible. Signs of pneumothorax were present on the right side. A Roentgenogram demonstrated 20 per cent collapse of the right lung but did not show air in the mediastinum. (No lateral view was taken). Very little dyspnea was noted. The patient had a low-grade

fever for six days, with no leucocytosis. She was given penicillin for three days, and had sedatives for pain. Hamman's sign remained positive for eight days. Roentgenographic examination on the seventh postoperative day showed moderate re-expansion of the right lung and a very evident mediastinal emphysema. All symptoms had disappeared by the tenth postoperative day when the patient left the hospital. There was never any subcutaneous emphysema. The pathological diagnosis of the excised thyroid gland was Riedel's struma with plasma cell infiltration.

Comment: A positive Roentgenogram is diagnostic of mediastinal emphysema, but a single negative report does not necessarily rule out its presence. It is submitted that many rather typical cases similar to this one may occur and are not reported. Milder degrees of the condition with little or no pain or with insufficient pressure to produce pneumothorax may often be unrecognized or disregarded.

Treatment.

A small amount of air in the mediastinum requires no active treatment but is a signal for vigilance. The air is usually absorbed in a few days and has no influence on the prognosis. Larger quantities of air necessitate measures for controlling the source and for providing an outlet for the air already accumulated. Diligent efforts to eliminate any respiratory obstruction must be instituted. For dyspnea or cyanosis inhalations of oxygen should be administered, but never under positive pressure if pulmonary interstitial emphysema from ruptured alveoli is a possibility. Artificial respiration is contraindicated. Both Keis[57] and Ackerman[86] mentioned that this tends to increase the mediastinal pressure and facilitate the production of pneumothorax. Pain may be alleviated by analgesics, and excessive coughing should be controlled. Emphysema in itself and unaccompanied by infection does not require antibiotics.

Signs of increasing mediastinal pressure are an indication for aspiration of air from the mediastinum. Incision through the suprasternal notch down to the trachea to permit escape of air through the superior aperture is commonly recommended. In tracheotomy and thyroidectomy cases, however, a recent neck wound is already present. It would seem logical to open the wound widely and even to insert a rubber catheter into the mediastinum if necessary. Barrie,[88] on the contrary,

advised removal of all drains and closure of the neck wound to prevent further entry of air by aspiration. This action would be unwarranted and even contraindicated if the emphysema were due to pulmonary interstitial emphysema. Suction applied to the incision at the base of the neck by the method of Tiegel[95] is considered by many as unnecessarily complex and of doubtful value. Aspiration of the mediastinum[79] can be performed most directly by the insertion of a needle attached to a syringe in the second or third interspace near the sternal margin and pointed toward the midline.

If tension pneumothorax is present immediate decompression is necessary. This may be accomplished by simple needle aspiration of the pleural cavity. If repeated aspirations fail to prevent reaccumulation of air continuous underwater drainage should be employed.

Subcutaneous emphysema, even if extensive, is not in itself an indication for other than conservative treatment in mediastinal emphysema. In fact those patients who display subcutaneous emphysema are, as a rule, less seriously ill than those who do not. Multiple skin incisions may facilitate the disappearance of air from the subcutaneous tissues.

SUMMARY.

Mediastinal emphysema occurring as a complication of operations on the neck is discussed. Eight cases complicating tracheotomy and three cases following thyroidectomy are presented.

The factors in the two operations tending to promote the condition appear to be analogous. An open neck wound exists in the presence of some degree of respiratory obstruction. The increased respiratory effort due to the inadequate airway causes exaggeration of the normally negative intramediastinal pressure and at the same time tends to increase the positive intrabronchial and intra-alveolar pressure.

Experimental evidence and clinical observation, as revealed in the literature, indicate that two different mechanisms operate in these cases to produce mediastinal emphysema. Air may enter the neck wound and be drawn along the fascial planes

into the mediastinum by negative intrathoracic pressure; or air under increased pressure may rupture overdistended pulmonary alveoli causing an interstitial emphysema of the lung with subsequent migration of the air along the perivascular sheaths to the hilus. Either mechanism, or possibly both together, may be the responsible agent in an individual case.

Mediastinal emphysema complicating tracheotomy and other neck operations probably occurs more frequently than a review of the literature would indicate. An active awareness of the possibility by the surgeon may be lacking or many cases of a mild type may be disregarded as of little consequence. Minimal degrees of mediastinal emphysema often may be unrecognized because no symptoms attributable to the condition exist. Routine post-operative Roentgenograms taken with the correct technique may be the decisive diagnostic measure.

The history of the subject suggests that the danger of mediastinal emphysema occurring with these operations may never be absent. Since the cause cannot be completely eradicated, the effects may be controlled or even surmounted by watchfulness and, when necessary, by prompt and appropriate intervention.

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A VARIATION IN TECHNIQUE OF FACIAL NERVE DECOMPRESSION.*

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INTRODUCTION.

Decompression of the facial nerve in its course through the temporal bone from the geniculate ganglion to the stylomastoid foramen is essential in the treatment of facial paralysis of otitic origin. Decompression of the vertical portion of the nerve from the horizontal semicircular canal to the stylomastoid foramen has been accepted by many as an adjunct in the treatment of Bell's palsy and has recently been advocated by Williams and associates[1] in the treatment of hemifacial spasm.

It was my belief that if facial nerve decompression and neurolysis could be performed with little discomfort, short hospitalization and postoperative convalescence, no change in hearing, and no other complications incident to the operation, it could be advised more freely to patients with Bell's palsy or hemifacial spasm. A technique was, therefore, devised by which the facial nerve could be decompressed from the stylomastoid foramen to or near the geniculate ganglion, without interruption of the ossicular chain, with preservation of hearing and with minimal trauma to the nerve and its blood supply.

REVIEW OF THE LITERATURE.

Historical: In 1812 Charles Bell[2] clearly showed that the VIIth cranial nerve is responsible for the motor innervation of the facial muscles and that it is not responsible for the pain of tic douloureux as was previously believed. In 1822 he[3] published a study on the anatomy and philosophy of

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expressions, separating voluntary and emotional responses of the face. Since his time peripheral facial paralysis not due to injury has been commonly known as Bell's palsy.

In 1879 Drobnik[4] united the peripheral end of the VIIth nerve to the central end of the external branch of the XIth nerve in a patient with facial paralysis with partial return of function. Numerous methods of anastomosis between the distal end of the VIIth nerve and the central end of various adjacent cranial nerves have since been suggested; in 1895 Ballance[5] anastomosed the VIIth to the XIth; in 1903, Korte[6] the VIIth to the XIIth, and in 1926, the VIIth to the IXth was reported in two patients by Watson-Williams[7]. Often a straight face in repose and some voluntary movement resulted, but these procedures were generally unsatisfactory because of associated movements and complete lack of emotional response of the facial musculature.

Love and Cannon[8] reviewed the cases of anastomosis of the VIIth to the XIth cranial nerves at the Mayo Clinic performed from 1926 to 1949. Most of the cases resulted from removal of VIIIth nerve neuromas and results of anastomosis in these were good in 75 per cent of the cases, but lack of emotional response and associated movements still existed.

Alt[9] was the first to advocate decompression in 1908 when he decompressed the facial nerve above and below an erosion in the facial canal produced by a cholesteatoma with recovery of the facial paralysis. Fourteen years later Ney[10] suggested repair within the temporal bone and in 1930 Bunnell[11] used a graft from the sural nerve to bridge a gap in the facial nerve in the temporal bone; but it is to the work of Ballance and Duel[12] set forth in their monograph that we owe our current concepts of intratemporal facial nerve surgery.

Anatomy—Localization of the lesion in facial paralysis is of utmost importance in any contemplated therapy. Theoretically, the lesion can be accurately localized; practically the lesion cannot be localized so specifically. The motor nucleus of the VIIth nerve lies deep in the lower portion of the pons; supranuclear pathways consist in *1.* crossed pyr-

amidal tract, *2.* uncrossed pyramidal tracts (supplying only the temporal branch fibers), *3.* extrapyramidal tracts from the emotional center in the thalamus, and *4.* connection with the Vth, IInd and VIIIth cranial nerves; thus, supranuclear or central facial paralysis involves only voluntary control of the lower portion of the face, and voluntary control of the forehead and complete emotional response are retained.[13] The facial nerve emerges from the lower border of the pons at the cerebellopontine angle, enters the internal auditory meatus with the VIIIth cranial nerve and courses anterolaterally to the geniculate ganglion. A lesion in this area produces loss of function of the VIIIth nerve as well as peripheral facial paralysis. The greater superficial petrosal nerve leaves the facial nerve at the geniculate ganglion, and geniculate lesions theoretically are localized by decreased lacrimation, loss of taste on the anterior two-thirds of the homolateral side of the tongue, hyperacusis and peripheral facial paralysis. The facial nerve leaves the geniculate ganglion entering the tympanic cavity just posterosuperiorly to the cochleariform process; entering the facial canal, it courses posteriorly for 8 mm., this portion being called the tympanic portion, where at a point just below the horizontal semicircular canal it turns inferiorly, still within the facial canal from which it makes its exit at the stylomastoid foramen 7 to 8 mm. below. The almost right angle bend below the horizontal canal is called the pyramidal portion. The stapedius branch of the nerve is given off from this portion, and the chorda tympani 2 to 3 mm. lower from the mastoid portion; thus, the presence or absence of hyperacusis and taste of the anterior two-thirds of the homolateral side of the tongue aids in localization of the lesion in peripheral facial paralysis.[14]

The blood supply of the facial nerve is from the internal auditory artery, the superior petrosal branch of the middle meningeal artery and the stylomastoid branch of the posterior auricular artery. Anastomoses occur above the pyramidal portion of the nerve but not below,[15] which possibly explains return of function of the chordae tympani and stapedius branches with persistent facial paralysis. After its exit from the stylomastoid foramen, the nerve turns abruptly forward dividing into its various branches deep to the superficial

lobe of the parotid gland; a lesion in this area (tumor, trauma) presents no problem in localization.

Etiology: Interruption of transmission of the neural impulses within the facial canal is generally conceded to be the mechanism responsible for Bell's palsy. Compression of the nerve without interruption of the axis cylinders does not cause failure of nerve conduction.[16] Hilger,[15] Kettel[17] and Sullivan[14] believed that the paralysis is due to local ischemia, which interrupts conduction and which may be accompanied by edema. This ischemia may be precipitated by exposure to cold, emotional factors and hereditary predisposition. Tickle[18] listed Bell's palsy as a toxic type of facial paralysis, and Collier[19] believed that many cases are viral in origin. There are a few patients who presumably have inadequate facial canals, as recurrent Bell's palsy is not infrequent.[17]

Pathology: The appearance of the facial nerve and the mastoid cells in patients with Bell's palsy who have undergone decompression was studied in 50 consecutive cases by Kettel.[20] Twenty per cent of the cases had necrosis of the mastoid cells, 36 per cent showed necrosis of the Fallopian canal, in two cases the nerve was exposed by necrosis, and in 56 per cent of the cases the nerve was grossly edematous in its vertical portion; microscopically there was no evidence of cellular infiltration. Cawthorne[21] reported constriction of the nerve at the stylomastoid foramen and bulging above in 12 of 14 patients who had undergone decompression.

Medical Treatment: Nonsurgical management of Bell's palsy has been directed toward relief of the ischemia and reestablishment of the blood supply by the early use of vasodilating drugs, sympathetic blocking drugs, and stellate ganglion block.[15,22] Recently, the use of adrenal cortical hormones in the early treatment of Bell's palsy has given encouraging results.[23] Support of the paralyzed muscles by the use of hooks, tape or intra-oral splints is generally advocated. Controversy exists concerning the use of galvanic or actual massage. Some writers [15,19] believe that the early use of galvanic massage retards fibrosis of the facial musculature whereas others[24] oppose galvanic stimulation because they believe that its use promotes fibrosis.

Selection of Cases of Bell's Palsy for Decompression: Bell's palsy may persist only a few days as a transient block or may progress to complete Wallerian degeneration. In approximately 80 per cent of patients with Bell's palsy spontaneous remission occurs.[11,24] The difficulty lies in determination of the 20 per cent who will need decompression. Once degeneration has occurred, recovery is never complete and is always marked by some mass movement and loss of function of the forehead muscles, whether recovery is spontaneous or follows decompression. Decompression of the facial nerve in Bell's palsy permits development of collateral circulation, reestablishment of circulation in the ischemic vessels and development of new vessels from the adjacent soft tissue.

Emphasis has been placed on loss of response to Faradic stimulation by Sullivan,[14] Cawthorne[21] and Tickle;[18] it is considered unreliable by Kettel[20] and Martin.[25] The presence of pain, completeness of the palsy, and rapidity of onset influence selection of cases for decompression. The advised time lag between onset of the paralysis and decompression varies from immediate decompression[14] to six months,[16] but averages eight to twelve weeks. All agree that the facial musculature must respond to galvanic stimulation if decompression is to be of value. Decompression is advocated enthusiastically by many[11,12,14,17,18,25] and with greater reluctance by others.[15,19] Use of electromyography may aid in detecting those patients who will recover spontaneously.[19,25]

Cryptogenic Hemifacial Spasm: This is an affection of the facial nerve and may be differentiated from facial tics (which are thought by some to be compulsion neuroses) by the following points: *1.* Hemifacial spasm involves only muscles innervated by the facial nerve; *2.* spasmodic facial tic is associated with tension relieved by performance and with anguish by suppression, and can be initiated and stopped at will whereas hemifacial spasm can not; *3.* hemifacial spasm occurs during sleep, but habit tics do not.[27] Cryptogenic hemifacial spasm must also be differentiated from hemifacial spasm secondary to other lesions of the facial nerve, as Bell's palsy and intracranial aneurysm.[28]

The etiology of cryptogenic hemifacial spasm is unknown.

Wartenberg[29] postulated that the facial nucleus contains two motor systems analogous to the pyramidal and lower motor neuron systems and that a lesion of the upper system produces hemifacial spasm and the lower Bell's palsy. Williams and associates[1] believed that hemifacial spasm is a lower motor neuron lesion, because a stroke does not affect the condition; hemifacial spasm is marked by weakness and contractures of the muscles, and known lesions of the lower motor neuron frequently produce hemifacial spasm. They found evidence of compression of the facial nerve in its vertical portion by fibrous tissue in four of eight patients with hemifacial spasm treated by decompression. Their immediate results were excellent, but the spasm recurred in two patients; permanent facial paralysis resulted in one, and there was a 50 per cent return of symptoms in another.

Patients with hemifacial spasm are miserable, embarrassed and handicapped. Heretofore treatment has been worthless, and there are few spontaneous remissions. If only partial or temporary relief can be obtained by decompression, it would seem worthwhile.

THE STUDY.

Decompression of the facial nerve by the postauricular route presents the disadvantages of difficulty in identification of the nerve at its exit from the stylomastoid foramen because of excessive bleeding, possible damage to the stylomastoid artery, postoperative discomfort and scarring, and an oblique view of the operative field. The usual endaural approach for decompression, in which the nerve is first identified just below the horizontal semicircular canal, or at a point just above the stylomastoid foramen by the blind removal of solid bone, presents the possibility of injury to the nerve before exposure is recognized.

The nerve is most readily identified in its tympanic course, where the Fallopian canal stands out in relief and adjacent landmarks are distinct; therefore, decompression without danger of trauma is best begun in this area, where the bony covering is quite thin. The nerve may be identified in this area by the following technique:

Technique: An open S-shaped endaural incision is made 2 to 3 mm. medial to the auricular cartilage beginning at seven o'clock in the right ear and five o'clock in the left ear. The incision is carried posteriorly and superiorly around the meatus through the periosteum to twelve o'clock and then extended superiorly superficial to the temporal fascia and

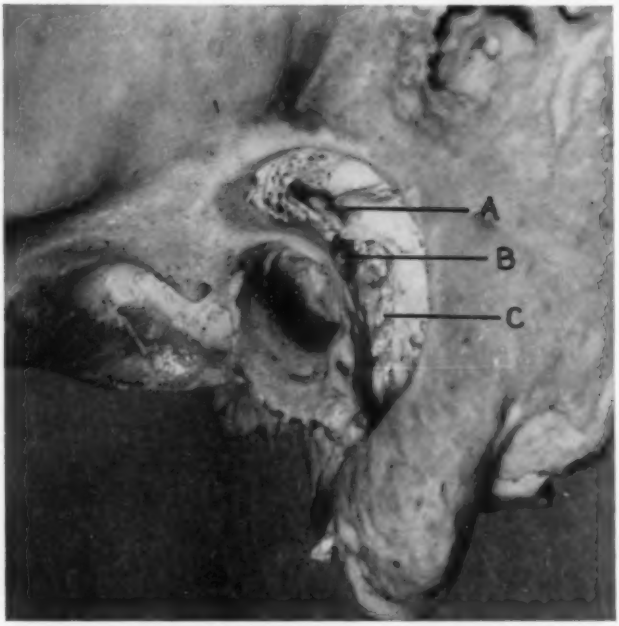

Fig. 1. Photograph of temporal bone.
(A) Short process of incus and horizontal semicular canal.
(B) Surgically created opening into tympanic cavity.
(C) Facial nerve.

muscle for a distance of 2 to 3 cm. The periosteum and superficial tissues are elevated from the mastoid cortex posteriorly, inferiorly and superiorly. A second incision is then made 1 to 2 mm. lateral to the first, sloping posteriorly beginning at the beginning of the first incision and extending to twelve o'clock, and forward 1 cm. so that a wedge-shaped

strip of skin, subcutaneous tissue and temporal fascia is removed (according to the technique of House[30]). A three-bladed endaural retractor is then inserted and the field exposed. Cortical bone is then removed with motor driven cutting burrs over the aditus, antrum and mastoid process, extending anteriorly until the antrum and short process of the incus are exposed and inferiorly and medially to the digastric ridge. The removal of bone is carried posteriorly to the sinus plate or less if the sinus lies far posteriorly. The superior and posterior osseous external auditory canal wall is then thinned with cutting burrs to expose the short process of the incus adequately; care is taken not to disturb the short process or the fossa incudis, and not to expose the incudomalleolar joint, The osseous canal is not lowered except in the patient with a forward lying sinus, in which case it is lowered only as much as needed for adequate exposure. After the canal has been thinned to a thickness of 1 to 2 mm., a hole is drilled with a 1 mm. polishing burr 1 to 1.5 mm. inferior to the lowermost extension of the short process of the incus, at a depth just superficial to the horizontal semicircular canal, and in an anteromedial direction paralleling the osseous external auditory canal (see Fig. 1). This enters the tympanic cavity at the level of and lateral to the stapes. This opening is then enlarged circumferentially, leaving a post of bone 1 to 1.5 mm. in width between the short process of the incus and the opening. The Fallopian canal is readily visualized through this opening in its course between the horizontal ampulla and the stapes, and in a roomy temporal bone the nerve may be seen up to the cochleariform process and geniculate ganglion. Visualization of the nerve in this area establishes the depth and general direction of the nerve and serves to orient its position. Removal of the dense bone overlying the Fallopian canal is then begun with cutting burrs sweeping from above downward. The operator uses as guides to the position of the nerve, the Fallopian canal itself above, and the junction of the digastric ridge and the tympanomastoid suture line inferiorly, which marks the position of the stylomastoid foramen. Under direct illumination and magnification bone is removed from the posterior and lateral surfaces of the Fallopian canal, slowly and with frequent irrigation to cool the area, until a bluish translucency, much as is seen in the

formation of the fenestra in the fenestration operation, is developed over the entire length of the nerve from the pyramidal portion above to the stylomastoid foramen below. The thin bone overlying the nerve is then carefully removed with the use of dental excavators and small curettes, and the stylomastoid foramen is enlarged posteriorly, care being taken not to injure the stylomastoid artery. The nerve is carefully inspected, and its sheath is slit along its posterior surface with a sharp myringotomy knife to avoid injury to the arteriolar trunk, which lies on the lateral surface of the nerve, as pointed out by Sullivan.[13]

The incision is then closed with interrupted 4-0 black silk sutures without drainage. The external auditory canal is packed tightly with selvage vaseline gauze strips, and a mastoid dressing is applied. Postoperatively, antibiotics are given routinely. The patient is discharged from the hospital on the third to fifth postoperative day and is seen in the office on the fifth postoperative day for removal of the sutures and the packing in the auditory canal. The dressing is left off.

This technique has been performed on five patients for Bell's palsy and three for cryptogenic hemifacial spasm. There have been no surgical injuries to the nerve incident to decompression, no postoperative complications, and no change in hearing in the operated ear. The period of absence from work has been minimal. The following cases are illustrative of results of decompression for Bell's palsy and for hemifacial spasm.

REPORT OF CASES.

Case 1: K. B., age 43 years, awoke with complete right facial paralysis on Nov. 15, 1952. This was accompanied by mild pain behind the ear. There was no history of chilling or other precipitating factors. When first seen on Jan. 18, 1953, complete medical, neurological, laboratory and Roentgenographic surveys revealed no abnormalities except complete right peripheral facial paralysis. On Jan. 23, 1953, approximately nine weeks from the onset of Bell's palsy, facial nerve decompression was done; the nerve appeared edematous throughout its vertical portion. The patient was discharged from the hospital on the fifth postoperative day. Examination three and one-half months postoperatively showed no change in hearing, a normal ear on otoscopy, and 75 per cent normal function of the right facial musculature (see Fig. 2).

Case 2: L. A., a white man, 47 years of age, was seen on Jan. 11, 1955, because of severe spasmodic contractions of the muscles of the right side

Fig. 2. (A) Photograph of patient with right facial paralysis.
(B) Photograph of patient one year following decompression.

Fig. 3. (A) Photograph of patient with hemifacial spasm in spasm.
(B) Photograph of patient not in spasm.

of the face, especially severe about the eye. He had no control over this symptom, which had been present for six years, and had become more severe in the past four months. Thorough examination of the ears, nose and throat revealed no abnormalities. Examination by a neurosurgeon disclosed no neurologic abnormality. On Feb. 9, 1955, the right facial nerve was decompressed from just below the cochleariform process to the stylomastoid foramen, and neurolysis was performed. There was no gross abnormality of the nerve. The patient was discharged from the hospital on the fifth postoperative day. The immediate postoperative result was excellent; there was no facial weakness, and the spasm was completely relieved for four months; however, six months postoperatively the spasm returned to 50 per cent of its previous severity (see Fig. 3).

SUMMARY.

In eight patients with Bell's palsy or hemifacial spasm, a variation in technique of facial nerve decompression has given better results than established techniques used in the past. By this method the facial nerve is decompressed from the stylomastoid foramen to or near the geniculate ganglion, without interruption of the ossicular chain, with preservation of hearing and with minimal trauma to the nerve and its blood supply. Although this series is much too small to justify conclusions, the absence of complications, lack of effect on the hearing, and minimal hospitalization and morbidity indicate that it may be a useful adjunct and worthy of trial in these patients.

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REPAIR OF CHRONIC CENTRAL PERFORATIONS OF THE TYMPANIC MEMBRANE: BY REPEATED ACID CAUTERY; BY SKIN GRAFTING.*

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Central perforations of the tympanic membrane are a common condition. It has been variously estimated that from one to six million people in the United States suffer from this condition.

Closure of one of these perforations is very gratifying to a patient. He may gain as much as 25 db of hearing. His middle ear and mastoid are protected from infected material in the ear canal (during bathing, swimming, etc.). He has less chance of an otitis media during an upper respiratory infection, and in some cases an annoying tinnitus is relieved.

I wish to discuss in this paper the office technique for closing perforations and also to describe a skin grafting procedure for closure of tympanic perforations which has interesting possibilities.

ANATOMY.

The pars tensa of the tympanic membrane is approximately 9×10 mm. in diameter. It is attached peripherally to the annulus tympanicus and centrally envelops the manubrium. The blood supply comes from the anterior tympanic and deep auricular branches of the internal maxillary artery and from the stylomastoid artery. There is rich anastamosis of the blood supply as it courses from Shrapnell's membrane along the posterior border of the manubrium and from around the periphery of the drum.

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HISTOLOGY.

The drum consists of four layers:

1. Squamous epithelium (external surface).

2. Radially arranged connective tissue from the manubrium to the annulus.

3. Annular connective tissue.

4. Middle ear mucosa (inner surface) of cuboidal to pseudostratified columnar epithelium.

PATHOLOGY.

Perforations are caused by trauma (burns, blasts, punctures, etc.) or infections (otitis media).

Regeneration of an intact tympanic membrane during healing is quite common; in fact, the cause of a perforation's becoming permanent is poorly understood. Dunlap and Schuknecht[1] have noted that in permanent perforations the outer squamous epithelium grows around the edge of the perforation to contact the inner mucosal layer; thus, they feel, acting as a limiting membrane on the middle connective tissue layer.

THERAPY AND REVIEW OF LITERATURE.

Before any chronic perforation can close, the limiting epithelium must be destroyed. Early therapy of perforations overlooked this and consisted merely of applying prostheses of various types: Banzer[2] in 1840 used pig's bladder membrane stretched over an ivory tube. Yearsley[3] in 1842 used a cotton wool pledget; Toynbee[4] in 1853 used Indian rubber and gutta percha. In 1850 Clark[5] used rubber, and in 1876 Blake[6] used paper. In 1887 Guaranowski[7] employed egg membrane; Pollack[8] in 1882, and Isaacs[9] in 1925 used collodion. More recently, in 1951, Pohlman[10] developed Koragel inserts.

Some investigators have noted that patching perforations occasionally leads to an actual closure: Barclay[11] in 1890 used paper discs. Other reports of closure by mere patching have

been made by: Nasiell[12] in 1934, and Steinmann[13] in 1945, using cellophane patches. In 1947 Unger[14] used gold foil and scarlet red ointment, and in 1951 Hall[15] reported closure of a fair percentage of perforations merely by applying a Yoshimo paper patch with 5 per cent celluloid in acetone as an adhesive.

The principle of cautery to destroy the limiting epithelium started with Roosa[16] in 1876, who used silver nitrate. Later Politzer[17] in 1895 also used silver nitrate, and Okuneff[18] in 1895 introduced trichloracetic acid. Joynt[19] in 1919 combined cautery with patching, and since then cautery with some form of patching has been used successfully by Stinson[20] (1941), Alexander[21] (1943), Fox[22] (1945), Ireland[23] (1946), Korkis[24] (1947), Folbre[25] (1947), Baron[26] (1948), Jordan[27] (1952), and Schrimpf[28] (1954).

The use of repeated cautery at roughly weekly intervals to prevent re-epithelization was used by Allport[29] (1916), Dunlap[30] (1917), Linn[31] (1944), Szpunar[32] (1947), Dunlap and Schuknecht[1] (1947), Adams[33] (1952), Derlacki[34] (1953), Peele[35] (1953) and Mullins[36] (1953). Derlacki[34] has noted that perforations can be closed by repeated cautery, which had failed to close by the cautery and patch method. This has also been our experience in our series of 65 perforations.

We feel that the cautery and patch method is successful where there is a fair blood supply, and destruction of the limiting epithelium is followed by rapid outgrowth of granulations, from the middle layer of the drum, which keep ahead of epithelization until opposing edges meet and seal with closure of the perforation.

Unfortunately, in many cases this exuberant granulation growth does not occur, and epithelization is able to circumvent and limit the healing edge of the perforation. This situation seems to arise when a drum is very scarred, and appears to have a poor blood supply.

METHOD.

Our technique is essentially the same as that of Linn[31] and Derlacki.[34] It consists of weekly cauterizations of the entire

margin of the perforation with 50 per cent trichloracetic acid. This is applied with a thin wire applicator tipped by a tightly wound cotton ball about one mm. in diameter. The wire should be malleable, as it may have to be bent to maneuver in tortuous canals. The applicator is dipped in acid, blotted gently on a kleenex and applied to the edge of the perforation and peripherally for a distance of about one mm. A white eschar forms over this area. A thin cotton patch is then packed down against the drum and kept moist with Neomycin drops five mg. cc., two drops twice daily. Therapy is continued until Politzer inflation and pneumo-massage of the drum with the Seigle otoscope demonstrates that the drum is completely intact.

Before closure is started the perforation is measured with a fenestrometer and an audiogram is usually done, and is repeated after patching the perforation with cigarette paper. The resulting hearing gain has been found to be a reliable index of the final hearing level after the closure.

OBSERVATIONS DURING CLOSURE ROUTINE.

1. The first few treatments are painful. To obtain better cooperation, especially in children, we place a cotton patch moistened with Bonain's solution (*i.e.*, equal parts of cocaine, menthol and phenol) against the drum for 10 minutes before treating the ear. After the first few treatments this is usually not necessary.

2. Some perforations, even very large ones, respond promptly and close in a few weeks; others, equally large, quickly show an active border, but growth is sluggish and any missed treatments usually stop growth advance. An active border is thick and slightly hyperemic; it bleeds easily and turns white with the slightest touch of trichloracetic acid, even as weak as a 5 per cent solution.

In some markedly scarred drums, especially those caused by burns, trichloracetic acid will not form a white eschar. This indicates a long and halting course in the closure, probably because of a poor blood supply. In these we apply acid heavily,

sometimes over the entire drum to destroy the scarred area and enlarge the perforation peripherally to a better blood supply. After months, an active border appears, and then narrowing of the perforation begins. It progresses haltingly, often with periods of temporary widening, and any missed treatments cause a loss of activity. Any attempt to change treatment, such as the use of a patch, or change to a weaker (5 to 10 per cent) trichloracetic acid has, in our hands, resulted in cessation of growth. Occasionally, after a very long time, these perforations have become very active and close rapidly. More frequently the final one-half to one-fourth mm. of the closure has been a prolonged process, with the perforation fluctuating in size and sometimes breaking open the week after it appeared closed. (All perforations should be followed closely for several weeks after they appear to be closed.)

3. Where therapy is discontinued before closure is achieved, scarring always gradually enlarged the perforation to almost its original size; however, where closure has occurred, this process merely thins the healed drum.

4. Besides Neomycin drops (5 mg. cc.) as a moistening agent for the cotton patch, we have used aqueous Furacin and 40 per cent alcohol. Others[1,31,34] have used Euthymol or 5 per cent urea in saline. Apparently no one drop is essential, but the cotton patch should not be allowed to dry. The functions of a moist cotton patch appear to be:

a. To prevent drying and crusting.

b. To provide a mildly stimulating irritation. Antisepsis is not too important; in fact the intervention of active infection has on several occasions activated sluggish or non-responding perforations to close rapidly. This has also been observed by Derlacki.[34]

c. If the ossicular chain is mobile, the moist cotton patch will cover the perforation and raise the hearing level. Our most faithful and appreciative patients have had bilateral perforations of this type. They will come back faithfully each week just for the hearing improvement which they receive by the cotton patch.

RESULTS.

From January, 1949, to May, 1954, we attempted to close 65 central perforations of the tympanic membrane. Total, marginal, and attic perforations, or the presence of cholesteatoma or squamous epithelium in the middle ear, attic, or mastoid were considered contra-indications to closure; however, no restrictions were made because of the size of the perforations, scarring, or adhesions to the promentory. All perforations had been present at least three months. Of the 65 perforations treated over this five-and--a-half-year period 57, or 88 per cent of them, were successfully closed. Four later reopened and of these, two quickly reclosed when treatment was resumed. A third case was closed by skin grafting, and the fourth case refused further treatment.

In a successful closure, how long a course of treatment may be expected? This table is a tabulation of all successful closures, showing the percentage closed at definite intervals:

	No. Closed	Per Cent Closed	Total Per Cent
0 to 10 weeks	26	46%	
2½ to 6 months	18	32%	78%
6 to 12 months	6	10%	88½
1 to 2 years	7	12%	100%

Thus, one-half of the closures occurred within 10 weeks and three-fourths of the successes occurred in the first six months, while 7 or 12 per cent took one to two years. We feel that several of our failures might have closed had they been as faithful as these seven cases.

ANALYSIS OF FAILURES.

An analysis of the eight failures shows that except for three (Nos. 6, 7 and 8), they were behaving in about the same way as did some of the similar perforations which later closed.

In our present state of experience we believe that with faithful return for treatment we would be perfectly justified in in-

forming a patient that his chances of eventual closure by this method are very good (approximately 90 per cent).

These results in the office treatment of perforated eardrums are comparable with those of other investigators using this or similar methods. We have in general been quite satisfied; however, there are two rather major shortcomings of this method:

1. Very young children cannot be treated satisfactorily because some cooperation by the patient is necessary.

2. If treatment is prolonged, some patients tend to become discouraged or are unable to return at the regular weekly intervals which are so important to success.

Case	No. of Treatments	Orig. Size in Mm.	Size When Last Seen	Remarks
1	20	3x2	2½x2	Patient discontinued treatment
2	21	4x4	2 mm.	Patient discontinued treatment
3	50	6x5	½x½	Moved away
4	52	6x5	½x½	Discharged as hopeless after one year
5	56	6x5	2x2	At one time was ¼ mm. across, then patch applied
6	71	5x4	2x2	Always irregular treatments because of work
7	80	3x3	1x1	Has since closed with skin graft
8	80	6x5	2x2	Still under treatment; fluctuation

It was in seeking a solution to these problems that a trial at skin grafting perforated eardrums was made. We have found that the idea, as in most instances, is not new. Berthold[37] in 1878 and 1890 reported on successful skin grafting procedures to close perforated eardrums. He cut very thick grafts the same size as the perforation and of a thickness so that when laid on the promontory they would fit the perforation like a plug. Two of these cases were followed 14 and 15 months respectively. Eli[38] in 1880 reported on skin grafting the middle ear promontory in chronic otitis media, evidently for the purpose of reducing the secreting surface. His results were poor, and no closures were effected. Roosa[39] also apparently tried this procedure. Tangeman[40] in 1883 reported a partial closure from skin grafting a dry perforation after roughing the margins.

Since this study was started, House[41] in 1953 reported four

successful closures by skin grafting. His method consisted of denuding the middle ear promontory, incising the drum remnant in several places so that it collapsed down into the middle ear, and applying a full thickness graft from the post auricular fold. His idea was to close off the middle ear cavity by partially obliterating it; however, in one case the graft eventually pulled off of the promontory and assumed a normal drum position. Zollner[12] in 1955 used essentially the same method without the relaxing incisions.

Between July, 1952, and July, 1955, we have performed 70 skin grafting procedures on 56 perforated eardrums. Fourteen of these procedures were regrafts after a first attempt had failed. Thirty-one, or 57 per cent of the grafts, were successful on the first attempt. Nine, or 64 per cent of the 14 regrafting procedures, were successful. Combining these, 40, or 71 per cent of the 56 perforations are now closed, and have been followed from three months to three years.

LENGTH OF FOLLOW-UP OF PERFORATIONS SUCCESSFULLY CLOSED BY SKIN GRAFTS.

Followed	No. of Cases
3 to 6 months	11
6 to 12 months	17
1 to 2 years	7
2 to 3 years	5
	40

FAILURES.

There were 30 grafts which are classified as failures. Twenty-six of these occurred *within six weeks after surgery*, because of slough, slipping or otherwise failure to close the perforation.

Four were late failures. These took primarily but later reopened.

The above statistics seem to indicate that once a tympanic perforation has been closed by a skin graft, it is fairly likely to endure.

While the overall percentage of successful graft closures is less than closures by the office method, gradual improvement in our technique has improved our expectancy of successful graft closures considerably.

COMPARISON OF EARLIER AND RECENT RESULTS.

70 Graft Closure Attempts.	
1st 10 cases	1 success
2nd 10 cases	4 successes
Last 10 cases	10 successes*

* Four of these last ten tympanic perforations were very large—approximately 8x7 mm. in diameter.

EVOLUTION OF OUR PROCEDURE.

The actual technique of this procedure has gone through considerable evolution, as we sought to better our results; however, the basic principles of the operation are fairly simple. The outer layer of squamous epithelium on the drum is dissected off with a special chalazion-type curette. It is peeled off as a layer (see Fig. 1). Skin from one of several sources is then trimmed to the appropriate size and placed on this raw bed. This method, when successful, produces a nearly normal free-lying eardrum. It differs from other methods in that no attempt is made to provide a bed for the central portion of the graft, which overlies the perforation. Berthold,[37] and more recently House[41] and Zollner,[42] used the promontory as a blood supply for the central portion of their grafts. Their methods, however, would be inclined to produce adhesions to the promontory and to obliterate, more or less, the middle ear. The question arises as to why the central portion of our grafts can survive without being in contact with any blood supply.

Pagett,[43] Conway[44] and others have studied the manner in which a graft "takes." For the first 48 to 72 hours the graft is nourished, and the waste products are disposed of by a "plasmic circulation," which they feel is largely extra cellular by imbibition. From 16 hours on, there is proliferation of endothelial buds from the host's blood vessels. At 65 to 72 hours, continuity of host's and graft's blood vessels has been noted,

and normal circulation in the graft is apparently largely complete and re-established by the eighth day. Preliminary fixation of the graft occurs within five hours by a fibrin clot. This clot gradually retracts and is invaded by leukocytes and phagocytes. On the fourth or fifth day fibroblasts begin to invade the clot, and organization is fairly complete by the tenth day.

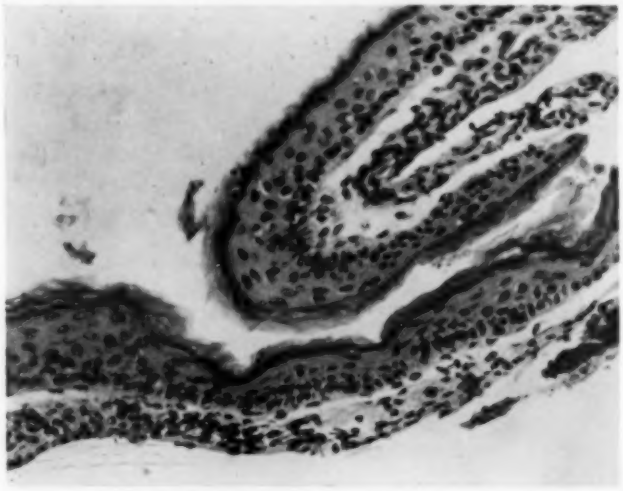

Fig. 1. Outer surface of tympanic membrane removed with a curette to make a bed for skin graft.

The above studies would suggest that eardrum grafts survive the first 2 to 3 days on "plasmic circulation," supposedly extravascular. McLaughlin,[45] however, feels that much of a graft's circulation in the first few days is intravascular, because a few of the many small severed blood vessels of the graft come in contact with a few of the many severed blood vessels of the bed. We believe that this concept of intravascular circulation in the fresh graft gives a much more satisfactory explanation for the survival of the central portion of our eardrum grafts which overlap the perforation. The following study supports this contention:

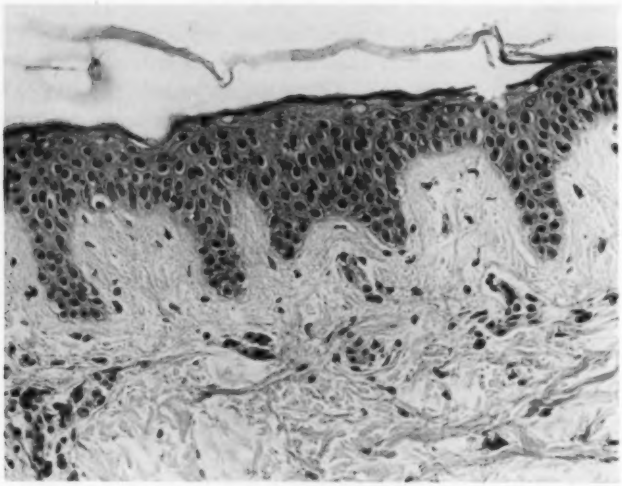

Fig. 2-A. Split thickness skin graft four hours after being placed on bed. Blood vessels are in spasm and contain no erythrocytes.

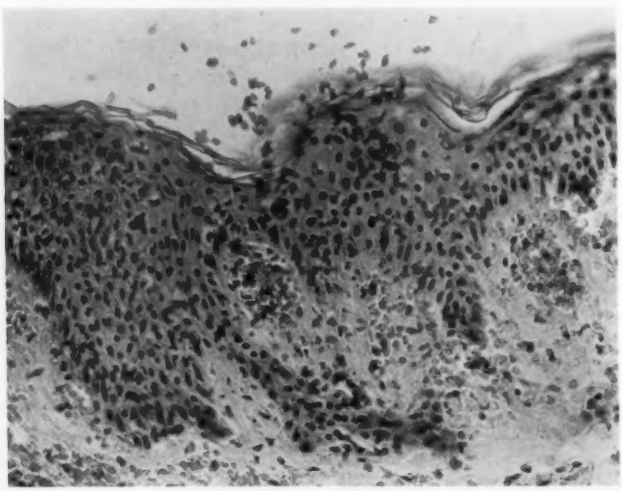

Fig. 2-B. Split thickness skin graft 24 hours after being placed on bed. Blood vessels are relaxed and dilated, and contain many erythrocytes.

Several split thickness skin grafts were cut and then laid back on their bed. After four hours microscopic sections showed an intense spasm of the graft's blood vessels, which squeezed out all erythrocytes (see Fig. 2). Microscopic sections of 24 hours' specimen showed, however, that the graft's blood vessels had relaxed, and all vascular spaces were filled with erythrocytes. This filling of the graft's blood vessels may have been from capillarity, or by continuity of graft and bed blood vessels. We believe it indicates some intravascular graft circulation.

In any case graft survival requires a good bed and a healthy graft. Atraumatic surgery is important, as is absence of infection. Good contact of graft and bed is also desirable, but we have found that too much pressure from packing may cause a slough of the central portion of the graft which has no underlying bed.

Many factors which affect results have been studied. It might be well to discuss them under separate headings.

1. Infection: We believe that several of our early failures were largely due to operating too soon (one or two weeks) after an otitis media. In spite of pre- and postoperative antibiotics, otitis media developed during the postoperative course of five cases. In each case, partial or total slough, or failure to seal the perforation resulted. At the present we feel that surgery should be delayed for at least one month after an otitis media. Sterile operating room technique is important. We also routinely use a sterile dressing technique for three weeks, and place the patients on antibiotics for one preoperative and six postoperative days.

Once a graft has healed, it may have surprising resistance to infection. One case has had at least four myringotomies done through the graft for acute suppurative otitis media. Each of these myringotomies healed after the infection had subsided.

2. Anesthesia: Local infiltration with 2 per cent Zylocaine, containing 1/50,000 Adrenalin. This may also be used topically on the middle ear mucosa. The addition of intravenous

sodium pentothal is usually necessary while the drum is being scraped.

3. Exposure: Exposure of the operative field is extremely important. The denuding of the drum, placing of the graft and packing must be done with exactness and can never be done blindly with success. In fact, adequate binocular visualization of all parts of the drum is essential, and magnification with the operating Loupe glasses extremely helpful. Occasionally, especially in children, the canal is both wide and straight, and the procedure can be done without an incision; however, we would attribute a fair number of our failures to working without enough exposure for binocular vision or magnification, because of a desire to avoid an incision in the ear canal.

Several methods of surgical exposure of the entire tympanic membrane are available:

a. A Shambaugh-type endaural incision and retractor can be used just as in an endaural mastoidectomy or fenestration. It is usually also necessary to cut a U-shaped flap from the posterior canal wall and to enlarge the bony canal posteriorly, superiorly and, occasionally, anteriorly. The flap has its interior incision about 3 mm. from the drum, and its pedicle on the floor of the canal. It is rolled down into the floor of the canal, where it is not in the way, and is replaced at the end of the operation.

b. In very tortuous canals where the anterior canal wall is the main visual obstruction, a U-shaped flap from the skin of the anterior wall can be made with the pedicle on the skin of the tragus. After the skin is out of the way the anterior wall bone is curetted or burred until the drum is visualized in its entirety.

c. A much simpler procedure, which we now use almost exclusively, is to curette the anterior canal wall with a very sharp curette, without elevating the skin. A sharp curette will cut through the skin and remove it without tearing it. The bony canal can then be enlarged and straightened by thinning the bone of the anterior wall. The new surface thus cre-

ated can then be covered with a primary or, later, secondary skin graft. This method will often give good exposure of the entire drum without an endaural incision and never requires more than a small T-incision at the superior margin of the canal.

4. Graft Bed: This is prepared as atraumatically as possible with 2 and 3 mm. chalazion-type curettes. They are angled 15 degrees forward or backward so as to maneuver in the canal. The outer squamous epithelial surface of the drum is peeled off, beginning at the edge of the perforation. Microscopic sections of the sheet of tissue removed have consistently shown that all of the squamous epithelium is removed, along with equal thickness of connective tissue (see Fig. 1). There are no rete pegs, which probably accounts for the fact that the entire specimen comes off as one layer. The entire layer removed measures from 0.15 to 0.2 mm., and the flat epithelium measures 0.05 to 0.08. There are no glands present in any section.

In our early cases the drum was denuded only 2 mm. from the edge of the perforation, and the graft was cut to approximately the same size. Now the bed and graft are made at least as large as the entire drum, and in very large perforations the denuding process is continued up onto the canal wall. This increase in the size of the bed decreases the chance of a slough. The increase in the size of the graft relative to the perforation makes it less likely that the graft will slip far enough to expose part of the perforation, or will be pushed through the perforation.

5. Cutting of Skin Graft: Four sources of skin for grafting of perforations have been tried. It is very important to cut these grafts just before they are used, so that they will not dry out. We feel that placing them in normal saline is injurious.

a. Split thickness skin graft from the inner surface of the upper arm (25 "takes," 9 sloughs). Miscroscopic sections show that this graft has very little hair and sweat or sebaceous glands. The skin is thin, and only a medium thickness graft is

available. Its tendency to curl is easily corrected by backing the graft with rayon or cigarette paper, using aureomycin ointment as an adhesive. The donor site is covered with surgical rayon, gauze and elastoplast. It heals consistently in ten days, when it is dressed for the first time. The danger of contamination and infection during bathing, etc., and the inconvenience of the dressing inherent in grafts from the thigh is not present here. The cosmetic effect is nicely hidden, because the donor site is on the inside of the upper arm.

b. Three-quarter thickness grafts from the mastoid area (22 "takes," 1 slough). These were all thick grafts. Microscopic sections show that this skin contains many sweat and sebaceous glands and quite a bit of hair. The graft should be taken from in front of the hairline. Several of our best results have long hairs growing from the drum. This skin has less tendency to curl and is easy to work with. The donor site is included in the ear dressing, and heals inconspicuously in ten days. We are now using this source almost exclusively.

c. Full thickness grafts from the post auricular fold (1 "take," 3 sloughs). House,[41] and Zollner,[42] used this source in their cases. The skin is very pliable and easy to work with. It has only a moderate tendency to curl and has softer hair and fewer sweat and sebaceous glands. We have not used this source extensively because Pagett[43] says, and we agree, that split thickness grafts "take" much more readily than full thickness grafts. There is certainly a good deal of additional trauma in trimming the fat from the under surface of full thickness grafts.

d. Pedicle flaps from the ear canal, both delayed and immediate (no "takes," 4 sloughs). There is considerable technical difficulty in swinging such a flap; also, a large flap necessarily has a small pedicle and any packing would, of course, crease the pedicle and cut off circulation. The skin, however, has a very fine texture and does not tend to curl. These poor results may be partially due to the extremely large perforations which were attempted by this method; also, sea sponge packing was used in all four flap cases. In six of the seven cases where sea sponge packing was used, slough occurred.

6. Graft Placement: If there is any tendency for the graft edges to curl, it is backed with surgical rayon, using aureomycin ointment as an adhesive. The graft is then cut to the appropriate measured size with a sharp knife. If it is to extend very far up onto the canal, the peripheral edges must be serrated with a sharp knife to allow for folding of the edges of the graft. The graft is then gently picked up with fenestration forceps and placed near the drum. It is then teased into position with forceps, fenestration picks, ring curettes, or mastoid searchers. After it is apparently in place the edges are momentarily rolled back with a fenestration pick to assure eversion and to see that all the perforation is well covered. The graft is then covered with cigarette paper and a cotton pack.

7. Packing: It is important to get good contact between graft and bed. With this in mind, we first tried tight vaseline gauze packs, or sea sponges. These tight packs had a tendency to push the graft through the perforation into the middle ear. We also developed a feeling that the part of the graft overlying the perforation was more likely to slough when a tight pack was applied (as mentioned before, a tight pack might obliterate the "plasmic circulation" to this portion of the graft).

Later we dispensed entirely with packing and depended upon the contracting of the underlying elastic clot to bring the graft into contact with the bed. On a few of these cases the graft slipped so as to expose part of the perforation, or failed to seal, and we have now settled on the method of packing used by Rosen[46] in the stapedial mobilization operation. After the graft is in place, it is covered with sterile cigarette paper strips, which extend up onto the canal, and very thin cotton balls are used to hold the graft gently in contact with the bed. After the first layer, aureomycin ointment is mixed with the cotton to protect from infection and to make the pack easier to remove.

8. Postoperative Routine: The patient is kept on antibiotics for six days. The packs are removed entirely on the seventh day. The cigarette paper and debris are wiped out, under

sterile precautions, on the fourteenth day. If raw areas are present on the canal, they are at this time covered with a secondary split thickness skin graft from the inner surface of the upper arm.[47]

The patient is Politzerized at one month.

We think it is a mistake to delay the complete cleansing of the canal and the Politzerization beyond the time specified. No harm can be done to a healthy pink graft and, if there is weakness, partial slough or failure to seal, it should be discovered as early as possible, as a regrafting procedure is best done as early as possible while the blood supply of the bed is still stimulated.

REGRAFTING PROCEDURES.

Fourteen of the failures were regrafted, with nine of these attempts leading to successful closure.

1. Four of the regrafting attempts were made in 1952. Two were done by split-thickness grafts from the arm, and two by rotation flaps from the skin of the ear canal. All failed.

2. The other ten regrafts were all done during 1955. Eight of these were successful, and the one failure had a third attempt at grafting, which was finally successful.

We have a general belief that regrafting of failures is perhaps more likely to succeed than the first graft, and think that regrafts should be done as soon as it is obvious that the first graft has failed. There is a great stimulation to the blood supply of the bed for a few weeks. It is probably best to remove the old graft entirely, freshen the bed, and apply an entirely new graft; however, three of our successful cases were done by a different method. The old graft was partially rolled back and the exposed bed freshened. A new graft was applied to this bed and laid against the rolled-back surface of the old graft. The edges of the two grafts were carefully everted and held in place with a soft cotton ball pack on cigarette paper.

ANALYSIS OF POSSIBLE CAUSES OF FAILURE.

In most of the 30 grafts which failed, it was believed that the factor causing the failure could be identified. Surprisingly enough, there did not seem to be any apparent correlation between the size of the perforation and either success or failure. The 56 perforations grafted varied from 3 × 3 mm. to 8 × 6 mm. The average size was approximately 4 × 5 mm. We consider that over a larger series of cases the smaller perforations should show a better chance of successful closure by skin grafting.

1. Four grafts "took" but did not seal in one quadrant:

a. Two of these were obviously from infection in the postoperative period.

b. The other two were in the same patient, and were ostensibly from blowing off the graft by Politzerization two weeks after surgery. We now wait one month before Politzerizing.

2. Five grafts "took" but slipped enough so that the perforation was not entirely covered.

a. Three of these were partly pushed into the middle ear, probably by a combination of too tight a pack and too small a graft.

b. In the other two cases the graft slipped enough to expose part of the perforation. No pack was used in these cases, which may have been responsible for the slipping.

3. Seventeen grafts partially or totally sloughed.

a. Five of these were obviously from infection.

b. In six, sea sponge packing was possibly responsible (as mentioned previously, six of the seven cases where sea sponge packing was used resulted in a slough).

c. In six cases there was no apparent cause for the slough.

4. Four grafts were late failures (the graft "took" but several months later, re-perforated). Two of these were caused by trauma, one by an otitis media, and one was apparently spontaneous.

LATE APPEARANCE OF GRAFTS.

The successful graft at first appears thick and hyperemic. It is sealed quite tightly at three weeks, and at six months is hard to remove. After the first year it gradually thins and becomes almost indistinguishable from a normal drum.

In one of our cases where no packing was used the graft "took," but slipped so as to leave a small slit of perforation still uncovered. Six months later this graft was removed and replaced with a fresh one, which was completely successful.

The old removed graft was a three-quarter thickness graft from the skin over the mastoid process. When it was removed with a curette the bed bled slightly. Microscopic sections through this graft showed that it was approximately the same (1.3 mm.) thickness as when it was cut six months earlier. There were still some sweat glands and a few hair follicles remaining. It was well vascularized, and there was almost no inflammatory reaction. The squamous epithelium was slightly flatter, but there were still rete pegs. At the periphery of the graft the squamous epithelium had grown around the edge and slightly underneath it, until it contacted and fused with the squamous epithelium of the surface of the drum. The area of attachment of the graft to the drum was quite vascular. There was mucosal lining which had grown across the under surface of the graft, where it overlay the perforation.

HEARING IN SUCCESSFUL GRAFT CLOSURES.

Twenty-five patients had hearing improvements of 10 to 30 db. Five failed to gain. In none was the hearing made worse. Ten patients were not tested. It was suspected that thick grafts would not give as good hearing as thinner ones. In two such cases the patient failed to make as much gain from grafting as from preoperative patching with cigarette paper; however, later as the grafts thinned, hearing came up to the expected level.

REPORT OF TYPICAL CASES.

1. Miss L. S., age 26, was seen in March, 1949, because of hearing difficulty all her life. Examination showed bilateral dry perforations; right 5x4 mm., left 4x3 mm. Closure routine was started. The left perforation

closed in eight treatments; the right in 11 treatments. Hearing gain right, 28 to 12 = 16 db; left 28 to 8 = 20 db.

Remark: This case illustrates how easily perforations sometimes close.

2. Mrs. M. J. G., age 27, was seen in February, 1954, complaining of an intermittent right otorrhea since childhood. Seven years previously she was advised to have a radical mastoidectomy. Examination showed a dry central perforation in the right tympanic membrane 5x4 mm. Mastoid X-rays showed no bone destruction. A patch increased the hearing for the speech frequencies in the right ear from a level of 23 to 3 db = 20 db gain. Closure treatment was started, using neomycin drops as the moistening agent. After the seventh treatment an active margin was noted except along the handle of the malleus. The entire margin was active after the eleventh treatment, at which time the perforation began to narrow slowly. Just after the thirteenth treatment the patient developed a severe painful external otitis and otitis media, with severe swelling. A pseudomonas aeruginosa was cultured from the drainage. Closure routine was stopped, and the patient put on aerosporin eardrops and codeine for pain. Ten days later when the infection subsided; no perforation could be demonstrated. Hearing gain, 23 to 3 db, exactly as predicted by patching. The drum now appears normal.

Remark: This case illustrates the beneficial effect of intercurrent infection on closures.

3. Mrs. W. A. C., age 31, was seen in March, 1952, complaining of bilateral otorrhea intermittently with colds since childhood. Examination showed bilateral dry perforations in both drums; right, 5x4 mm.; left, 6x5 mm. The left drum was very scarred and the malleus handle was protruding into the perforation. Bone conduction was normal. Patching caused a hearing gain of 25 to 5 = 20 db, right ear; and 32 to 8 = 24 db, left ear. Closure routine was started, using neomycin drops. Over the next two years the patient received 90 closure treatments in the left ear and 72 in the right ear. During the first year, these treatments were broken up by an attempt at patching, a trial of weaker (10 per cent) trichloracetic acid, and finally an attempt at skin grafting the perforations. All failed. It was noted that missed treatments caused the perforation edges to become inactive. Eleven months after treatment was started, the perforations measured: right, 5x4 mm.; left, 4x3 mm. After this time, there was no variation from the usual weekly painting with 50 per cent trichloracetic acid, cotton patch and neomycin drops. The right perforation was closed after 20 months and 72 treatments; the left closed after 24 months and 90 treatments. Hearing gain: right, 25 to 2=23 db; left, 32 to 17=15 db.

Remark: This case shows how prolonged a successful closure can be. After one year of treatment these perforations were almost as large as they were before treatment was started; yet both were successfully closed. Few patients would return this faithfully.

4. Miss W. K., age 12, was seen August, 1951, complaining of intermittent otorrhea all her life. Examination showed a draining right ear with a 3x3 mm. central perforation of the right tympanic membrane. Culture revealed a pseudomonas aeruginosa infection, which cleared on neomycin drops. The patient then had 80 weekly treatments for closure, during which time the perforation fluctuated in size, on one occasion being only one-half mm. in diameter. She then became discouraged and discontinued treatment in July, 1953. When next seen in June, 1955, the perforation was as large as before treatment was started. On June 21, 1955, a split thickness graft from her arm was used to cover the perforation. It "took"

well and an audiogram three months later showed a hearing gain of 25 to 15=10 db. It is hoped that this will continue to improve as the graft thins.

5. Mrs. W. R. W., age 19, was seen June 11, 1954. Examination showed a dry 6x6 mm. central perforation of the right tympanic membrane. On June 24, 1954, via an endaural incision, the perforation was grafted with a split thickness graft from the upper arm. The graft "took" well and is intact 15 months later. It has thinned to approximately the same thickness as the rest of the drum. Hearing gain, 17 to 7=10 db.

CONCLUSION.

While one-half of these successful skin graft closures have been followed less than nine months, successful graft closures have been followed two and three years. So far, only a very few of the procedures have become a failure later than six weeks postoperatively. None, so far, have developed as failures later than four months postoperatively. It is hoped, therefore, that the long term results reported for these skin graft closures will continue to hold up. We realize that the office method of closing tympanic perforations is a well-proven procedure, while much has still to be learned about skin graft closures; however, our results with skin grafting are encouraging, and the advantages of grafting are considerable in dealing with children and where a prolonged course of office treatments is anticipated. We feel justified in recommending this procedure to any patient when weekly office cautery has not made progress toward closure within ten weeks. We also feel that in the event a graft is unsuccessful, a second, and if necessary a third graft can be advised.

SUMMARY.

Closure of 57 of 65 central eardrum perforations by weekly cautery with trichloracetic acid is reported.

Closure of 40 of 56 perforations by skin grafting is reported.

The literature on this subject, and factors affecting results are discussed.

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SEVENTH INTERNATIONAL CANCER CONGRESS—LONDON, ENGLAND.

The Seventh International Cancer Congress will be held in London, England, July 6-12, 1958, under the Presidency of Sir Stanford Cade. Congress headquarters will be The Royal Festival Hall. This Congress is sponsored by the International Union Against Cancer.

There will be two main sessions of the Congress: A. Experimental; B. Clinical and Cancer Control. Special emphasis ill be placed on hormones and cancer, chemotherapy, carcinogenesis and cancer of the lung.

Papers will be considered only if submitted with an accompanying abstract (not over 200 words) before October, 1957, and if dealing with new and unpublished work.

The registration fee for the Congress will be £10 (ten pounds) or $30, and the latest date for registration without late fee will be January 1, 1958.

Registration forms and a preliminary program will be available early in 1957 on application to The Secretary General, Seventh International Cancer Congress, 45 Lincoln's Inn Fields, London, W. C. 2, England.

EDUCATIONAL GUIDANCE FOR PARENTS OF YOUNG DEAF CHILDREN.

EDGAR L. LOWELL, Ph.D.,
and
GEORGINA RUSHFORD, M.A.,
Los Angeles, Calif.

Ten years ago THE LARYNGOSCOPE published an article from the John Tracy Clinic entitled, "What the Otologist Should Know About the Educational Problems of Deafness." To quote from the article:

> "To sum it all up the doctor should be able to offer constructive advice to the patient he cannot help medically. He should have at hand concise, accurate and definite information as to what may be done for such a patient—child or adult. He should know where the patient may go for help. He should know what local and national organizations offer facilities for the hard of hearing and the deaf.*"

A decade later we are able to report how well this need is being filled. As part of a larger study carried on to evaluate the success of the Correspondence Course distributed by the John Tracy Clinic, we have collected factual information on the kind and amount of educational advice given to parents of young deaf children.

At the John Tracy Clinic, where we have been primarily concerned with educational guidance, we are constantly impressed with the importance of the educational information given by the doctor. At the crucial time of interpreting the diagnosis of hearing loss to parents, prompt and positive suggestions do much for the future adjustment of the family and child.

The main intent of the larger study was to evaluate the Cor-

* Harriet Montague: What the Otologist Should Know About the Educational Problems of Deafness. The Laryngoscope, Vol. 56, No. 6, June, 1946.

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respondence Course which the Clinic offers to parents of young deaf children all over the world. The course was first sent out in 1943, and during the 13 years since then more than 7,000 families have been enrolled for these lessons, from approximately 52 different countries. The course covers one year of work that any untrained parent can do at home without supervision except that supplied by mail from the Clinic. The program is explained in simple language, and it is necessarily flexible since the children range in age from 22 months to 5 years. The idea is to provide suggestions easily understood and easily followed, to give the parents something constructive to do to help their deaf children, and to give the children themselves something constructive to do to utilize their energies and natural abilities, thus putting them into communication with the world around them.

The material on which these figures are based comes from the first 5,000 families enrolled in the course. The course has been sent to all parts of the United States and has had a geographical distribution approximately the same as the total population of the United States.

At the time of parent application 854 of the children had not yet had an accurate evaluation of the extent of their hearing loss; 1504 had been classified as hard of hearing, with average losses up to 75 decibels through the speech range; and 2645 had been classified as severely or profoundly deaf with losses greater than 70 decibels. The majority of these parents became aware of their children's deafness through the child's failure to respond to sound. Specifically, in 40 per cent of the cases it was failure to respond to the parent's voice, and in 23 per cent of the cases failure to hear loud environmental sounds. Failure to develop speech at the normal time accounted for 26 per cent, and the remaining 6 per cent became aware of the loss only at the time of the doctor's diagnosis.

The importance of the doctor's role in providing such information is emphasized by the fact that 92 per cent of the parents in this study had no previous knowledge about deafness and the education of the deaf. Seventy-one per cent of this parent-group indicated they had received advice from the

physician concerning their deaf child's education. Many years ago the importance of beginning the specialized education required by a deaf child at the earliest possible moment, was not recognized. The fact that the average age of the children in this sample was 3½ years indicates the medical profession is now cognizant of this trend and is working to support it.

Seventy-seven per cent of the doctors discussed the teaching of speech with the family. Some few parents still erroneously believe that the manual method, that is, finger spelling and signs, is the only avenue of communication possible for the deaf person. To know that a child can acquire language and speech is important in helping a family adjust to the situation.

The questionnaire on which most of this information was based did not specify whether the doctor who provided the educational information was an ear specialist, a general practitioner, or a pediatrician; in fact, only 715 of the families reported they had received specialist treatment at the time of application for the course. This evaluation of the medical recognition of educational problems of deafness reflects knowledge on the part of the entire profession and not specifically on the part of otologists. We feel this is a great compliment to the entire profession.

It is unreasonable to expect the doctors to provide prolonged guidance; however, they are making this possible by putting parents in touch with organizations specifically designed to provide educational follow-up. Seventy-one per cent of the doctors knew the names of the schools for the deaf nearest where the family lived and referred them to other organizations offering help. Sixty-two per cent gave the families specific help with additional sources of information. This examination of the Clinic's records indicates that 40 per cent of all applicants for the Correspondence Course were referred by the medical profession.

Further evidence of the close cooperation between the medical and educational fields comes from the fact that only 4 per cent of the doctors were reported as still using the old-fashioned and incorrect term, "deaf and dumb" in discussing

children who are only auditorily handicapped. This antiquated term has unpleasant and misleading connotations for the parents and the general public.

Totally, these figures indicate that doctors have been doing an excellent job with a difficult but essential educational problem.

A SPECIAL POSTGRADUATE COURSE.

An introduction to fundamentals of reconstructive surgery of the nasal septum and external pyramid will be presented by Yale University School of Medicine Department of Surgery, Section of Otolaryngology, in cooperation with The American Rhinologic Society and Grace-New Haven Community Hospital, May 11 - 18, 1957.

The course will be given under the guest direction of Dr. Maurice H. Cottle. It will consist of didactic lectures, case presentations, cadaver dissections, and observation of operations. All sessions will be held at the Yale-New Haven Medical Center, New Haven, Connecticut, U. S. A.

Enrollment will be limited. Applicants must be diplomates of the American Board of Otolaryngology, or its equivalent for doctors from other countries. Scholarship assistance is available to a limited number of foreign applicants.

For further information write to Assistant Dean of Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut, U.S.A.

HEARING LOSSES IN CLEFT PALATE CASES.†

The Incidence, Type and Significance.

MAURICE H. MILLER, M.A.

New York, N. Y.

PART I. NATURE OF THE STUDY.

Each patient with a cleft palate referred to the Speech and Hearing Clinic at Lenox Hill Hospital is given a complete pure tone audiometric test as part of the preliminary workup before speech therapy is begun. The susceptibility of individuals with this organic involvement to various types of upper respiratory conditions with its frequent effect upon the hearing mechanism has convinced us of the need for a careful investigation of the hearing of all cleft palate cases.

The audiometer used in conducting these tests was a Sonotone AE21 model. The testing was done in a sound treated room where quiet conditions prevailed throughout the course of the study. Threshold was established at the following frequencies: 125 c.p.s., 250 c.p.s., 500 c.p.s., 1000 c.p.s., 2000 c.p.s., 3000 c.p.s., 4000 c.p.s., 8000 c.p.s., 10,000 c.p.s., and 12,000 c.p.s. Whenever a loss of 25 db or greater was found by our air conduction tests, a bone conduction test on the affected ear or ears was performed. The definition of a hearing loss used in this study is a loss of 30 db or greater at one or more of the frequencies tested. We have chosen this intensity level because in many cases young children do not respond to pure tones the instant they hear them but wait until the sounds are clearly audible before giving evidence of having heard them. Thirty-five cleft palate cases were used in this study, ranging in ages from three to 23 years of age. As to the validity of audiometric tests on children three to five years of age in this study, we are convinced that after elicit-

† Research conducted in conjunction with clinical work at Lenox Hill Hospital and submitted as part of the requirements for the Seminar in Speech Pathology at Brooklyn College under the supervision of Dr. Robert W. West and Dr. John K. Duffy.

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ing the cooperation of the child it is possible to perform an accurate pure tone audiometric test on these children as shown by the consistency of results found in our retests of children in this age group.

PART II. FINDINGS.

The type of hearing loss found in the cleft palate cases tested is predominantly conductive; that is, a loss caused by some impairment of the sound conducting mechanism responsible for the transmission of sound waves to the inner ear. Although none of the cases tested had pure preceptive losses, there were five mixed losses found among the group. One was a child with congenital atresia of the right ear, and our hearing test showed a severe conduction loss (60-65 db) throughout the range tested with slight nerve involvement. Three cases of mixed loss showed a history of chronic suppurative otitis media in which the infection may have spread to the inner ear causing some nerve involvement. Another case having a mixed loss showed nerve involvement principally at 4000 c.p.c. with the remainder of the loss conductive in nature. This patient sustained possible traumatic injury to the head.

Of the 19 cases of conduction hearing losses found, there were 12 instances of conduction losses affecting the high frequencies only, and probably due to the presence of lymphoid tissue in the naso-pharynx. There were seven cases in which there was a conduction hearing loss throughout the range tested. The results of the study are summarized in the Table on p. 1494.

It will be noted that in Part III of the table, which gives a breakdown of the range of frequencies involved in the 19 cases of conduction hearing losses found, no percentages are listed, and the total number of the cases is 21, rather than 19. This is because some of the cases of bilateral hearing losses showed a different type of conduction loss in each ear and were, therefore, mentioned in two of the categories. For example, in the case of a patient having a high frequency conduction loss in one ear and a loss throughout the range in the

other, the first type of conduction loss was listed in Part IIIa, the second in Part IIId.

Classification	Number of Cases	Per Cent of Total Cases
Number of cleft palate cases tested	35	100%
I. Of the total number of cases tested, those having:		
a. no hearing loss	16	45.71%
b. some hearing loss	19	54.29%
		Per Cent of 19 Cases Having Some Loss
II. Of those cases having some hearing loss, those with:		
a. pure conductive loss	14	73.68%
b. pure preceptive loss	0	0%
c. mixed loss	5	26.32%
III. Of those cases having pure conduction losses, those with:		
a. high frequency conduction loss in one or more ears	12	
b. low frequency conduction loss in one or both ears	1	
c. conduction loss in one or both ears at very low and very high frequencies only (128 c.p.s., 256 c.p.s., 10,000 c.p.s., 12,000 c.p.s.)	1	
d. conduction loss throughout range tested	7	

PART III. SIGNIFICANCE.

The high incidence of conduction losses of hearing in cleft palate cases may be due to the presence of the cleft in the hard or soft palate which permits the entrance of organisms producing upper respiratory infections, chronic colds and influenza, otitis media, etc. In addition to this factor, the incomplete closure of the naso-pharyngeal port caused by an unusually short velum which does not make contact with the superior pharyngeal constrictor is another means by which the organisms producing upper respiratory conditions can gain access to the body.

In a large number of cleft palate cases, the hearing loss appears to be caused by the presence of prolific lymphoid tissue in the naso-pharynx which occludes the mouth of the Eustachian tube and interferes with the proper ventilation of the middle ear. The presence of this condition has been determined by direct and indirect naso-pharyngoscopy on some of our cleft palate cases having conductive losses of hearing. The removal of this lymphoid tissue is frequently contra-indicated in cleft palate cases (as is the removal of the tonsils) because any tissue in the area of the oro- or naso-pharynx seems to help the child to achieve the closure of the naso-pharyngeal port so necessary for the development of intelligible speech.

Dr. Fred S. Dunn, Chief of the Oral Surgery Division at Lenox Hill Hospital, suggests as an explanation for the high incidence of hearing losses among cleft palate children, the practices of some cleft palate surgeons who, during an operation to close the soft palate, frequently produce scarring or distortion of the tissues at the naso-pharyngeal orifice of the Eustachian tube interfering with its normal function.

The type of conductive loss found most frequently in cleft palate cases is one which affects the very high frequencies first (8000 c.p.s. to 12,000 c.p.s.) and then, if the cause of the hearing loss is not discovered and proper medical care administered, the entire range, resulting in the reduced acuity for all speech sounds characteristic of advanced conductive losses. The frequency of high tone losses with no nerve involvement found in these cases tends to support the findings of Crowe[1,2] who stated that high frequency losses can be purely obstructive in nature.

This type of conduction loss, in its initial stages, does not affect the ability of the patient to comprehend speech because the frequencies affected are above the range important for the understanding of speech. Its significance lies in the fact that it is frequently the precursor of a more serious conductive loss and indicates the necessity of an immediate ear, nose and throat examination to determine the cause of the condition.

(We have found that conductive losses in children who do not have a cleft palate also tend to affect the high frequencies first, and then, if proper medical care is not administered the entire range of hearing.)

Because this type of conduction loss affects frequencies above the range important for understanding speech, speech reception tests such as the fading numbers test currently used in many public school systems and group pure tone audiometric tests and screening tests using frequencies only in the speech range will not be successful in discovering this incipient type of conductive loss at the time when a proper otological check-ups might arrest the condition. Only a complete, individually administered, pure tone audiometric test which tests frequencies above and below the speech range (125 c.p.s., 250 c.p.s., 10,000 c.p.s., and 12,000 c.p.s.) will detect this type of conductive loss. The public school systems, the hospital and university speech and hearing clinic, and all agencies working with large numbers of children should recognize the diagnostic significance of this prodromal conductive loss in cleft palate children and provide for adequate medical treatment through the services of a competent otologist. Proper medical treatment will frequently be successful in stabilizing and even eliminating the hearing loss and certainly in preventing later deafness in adulthood.

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In Memoriam

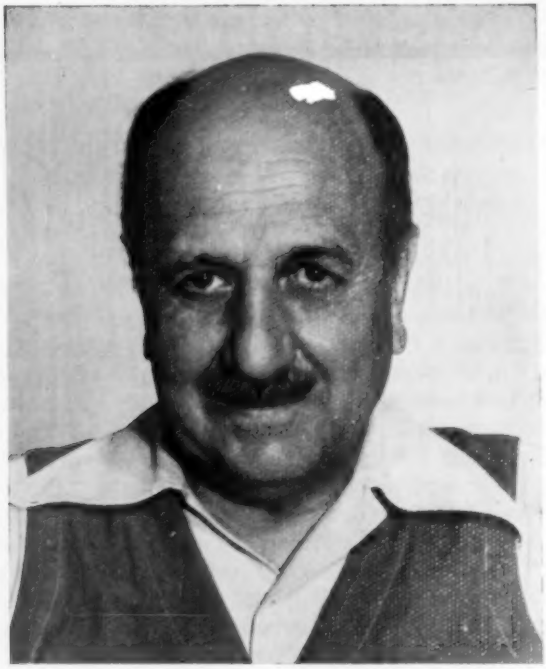
ISAAC HAMPSHUR JONES, M.A., M.D.,

1881-1956

Isaac H. Jones was born in Philadelphia in 1881. He attended Amherst College, from which he graduated Phi Beta Kappa. In 1906 he received his M.D. degree at the University of Pennsylvania School of Medicine, with election to Alpha Omega Alpha. In his early years of otologic practice he came under the spell of Robert Barany, whose "epoch-making" work on vestibular nystagmus, a short time before, had focussed the attention of otologists the world over. A new field was opened up, and Isaac Jones entered it with enthusiasm. In Philadelphia he had as guide and counsellor, Dr. B. Alexander Randall in otology, Drs. Charles K. Mills and T. H. Weisenburg as neurologists, and Drs. Lewis Fisher and Seth A. Brumm as fellow-workers.

By 1916 he had become a recognized leader in the otologic quest, and was invited to address the Academy of Medicine in Washington, D. C., on the ear and aviation. In his audience was Col. Theodore Charles Lyster. Later, after this country entered World War I, Dr. Lyster brought Dr. Jones into Army service and introduced him to his uncle, Major-General William C. Gorgas. In May, 1917, Lyster, Jones and Eugene R. Lewis formulated the "Physical Examination for Flying," which was made official for the Army as A.G.O. 609.

Under Col. Lyster, now Chief Surgeon of Aviation Section, Signal Corps, United States Army, Drs. Jones and Lewis were appointed to organize boards of medical exam-



iners of candidates for commissions in this new service. The two men travelled from one recruiting center to another, from the Atlantic seaboard to the Pacific coast, setting into activity 35 medical aviation boards.

Overseas in 1917 and 1918, he had intimate contacts with British, French and American flyers, as medical counsellor, observer and—pupil. Again in France, in 1919, at the Versailles Peace Treaty, Col. Jones was appointed medical advisor for aviation in the American delegation, and was one of the two American members of the committee which drafted the International Requirements for Commercial Fly-

ing. In recognition of his services the Government of France created him *Chevalier de la Légion d'Honneur.*

In the interval between his periods of overseas service he edited the *Air Service Medical,* with the collaboration of Frank L. Dennis, William F. Patten and Eugene R. Lewis; he also served for a time as editor of *Aviation Medicine.*

His *magnum opus,* "Equilibrium and Vertigo", the fruit of his clinical work prior to the war, and dedicated to his three mentors, Drs. Mills, Randall and Weisenburg, was published in 1918. In the preface he gives abundant credit to his collaborators, Lewis Fisher, Eugene R. Lewis, George E. Shambaugh and others, as well as a gracious tribute to his wife's assistance. (Dr. Fisher, named as co-author, wrote the section "An Analysis of Pathologic Cases", an account of clinical findings and their interpretation, in cases under observation from 1914 till 1917.)

By a happy circumstance (may we not call it a "fortune of war?") he was able to make an eleventh hour addition to the book's already rich contents, namely: Chapter III—"The Ear and Aviation". This brought formal acceptance by the Army, as shown on the title-page: "Adopted as Standard for Medical Division, Signal Corps, Aviation Section, by Surgeon General, and Chief Signal Officer, U. S. Army." This book may, in future years, prove to be a treasury of material for students of the history of medicine. (The same, in lesser degree, is to be said of his "Flying Vistas", published 19 years later.)

Leaving Philadelphia in 1920, he began a new practice in Los Angeles in association with Drs. T. C. Lyster, Samuel D. Ingham and Eugene R. Lewis and soon gained prominence in his field.

A common interest in acoustics brought together Prof. Vern O. Knudsen of the Department of Physics in the University of California at Los Angeles and Isaac Jones, clinician-otologist. Their joint labors resulted in a series

of articles on hearing problems, the creation of an early audiometer and of an experimental hearing-aid. He was appointed Research Fellow in Zoology at the University; Dr. Walter P. Covell and he conducted animal experimentation to determine the effects of vitamin deficiencies in rats—for example, changes in general nutrition and the degeneration of spiral ganglion cells resulting from withholding of yeast from the diets.

In another "Venture to the Interior" (to borrow a title from Laurens Van der Post) Drs. Ingham, I. Leon Myers and Jones studied in slow-motion pictures, the actions of cats dropped, with feet up, from a height of five feet to a mattress—normal cats; others with surgical lesions of the labyrinth and the cerebellum. Normal animals turned in the air in the first two feet of the fall and landed on their feet. Those with vestibular lesions fell without showing any tendency to right themselves. Dr. Ingham writes that "the results clearly demonstrate the major importance of vestibular function in the control of the cats' marvelously coordinated movements."

The significance of this study is shown in a section of Dr. Jones' last book, "Flying Vistas, the Human Being as Seen Through the Eyes of the Flight Surgeon," published in 1937. The volume tells the story of Aviation in World War I, and is dedicated to Gen. Theodore Charles Lyster. It was the duty of Drs. Lyster and Jones to organize the new Aviation Section, as already told. In the book we follow the course of its growth and the experiences of the two men in this country and in France. In its earlier chapters "it attempts to explain some of the mysteries of the human body, including the difficult things about the special senses . . . in plain English." In the chapter on Blind Flying, Dr. Jones sets forth the lessons of the "cat" experiments and their application "when we consider the illusions that come from the ears . . . when we are flying blind." The author's style is light-hearted, revealing and sensitive. The history-making

achievements of the Aviation Section are told, both gaily and with pathos. Any present-day flyer will do well to read this book, which won high praise from officials of the Aviation Corps. Of the Flight Surgeons of 1917-18 Dr. Jones was the first to receive a commission. In the words of a fellow-officer, "no one is better qualified to tell the tale of those thrilling days."

Dr. Jones was a prolific writer. Of his articles on acoustics many were the fruit of his association with Prof. Knudsen. His work on vestibular problems resulted in many contributions of weight, of which one in particular is chosen for special mention here—"Vestibular Experiences". It contains a detailed report of the case of a boy of 15 with "complete vestibular block . . . perfect auditory function and no vestibular function;" he and Dr. William G. Spiller had presented their findings in this case before the American Neurological Society* and he later reported it to the American Laryngological, Rhinological and Otological Society**: "Dr. Spiller . . . for the first time revealed the Central Vestibular pathways," a demonstration of the vestibular tract in the brain-stem which "was made possible by a sequence of events that may never happen again." This article includes also an account of vestibular damage from streptomycin; the discussion which followed its reading is unique in that one of the participants, Dr. Samuel D. Ingham, Dr. Jones' close friend of many years, gave a neurologist's review of his own off-balance experience, being himself a victim.

Nutrition and Related Problems was the subject of many articles, including a long series of editorials published annually in THE LARYNGOSCOPE. In most of these essays he had as co-workers Eugene R. Lewis and others, including Leland G. Hunnicutt, Jerome A. Hilger and the writer of

*Isaac H. Jones, M. D., and Wm. G. Spiller, M. D.: "The Central Tracts of the Nervous Vestibularis." Brain, Sept., 1925.

**Isaac H. Jones, M. D.: "Vestibular Experiences." Trans. of Amer. Laryngol., Rhinol. and Otol. Soc., Inc., 1949.

this obituary, who had the privilege of taking part in the work through a period of 15 years. It was his delight to inject here and there, when an opportunity came, a witticism or a timely quotation from an old-time author. Always, simple English was his goal; "let us make it shorter and so plain that A or B will get the meaning." His appraisal of material was sound. Those who worked with him were the richer for so doing.

In 1922 Dr. Jones enlisted the interest of other Los Angeles otologists and brought Prof. Robert Barany to this city for a course of lectures. (He often said whimsically: "Eleven were invited from other States, and of these 13 came.") Similar courses followed in the ensuing years. In 1928 the group took a long step forward in the formation of the Research Study Club of Los Angeles. An early major activity was a series of lectures, given by local otologists, neurologists and neuro-surgeons, and later combined in one volume, "Studies of the VIII Nerve, by the Research Study Club". In 1931 began the Midwinter Clinical Course, the first gathering being for otologists. Its success was so inspiring that for the following Convention in 1932 the ophthalmologists, led by Dr. A. Ray Irvine, joined forces with their colleagues. Thus came into being the annual Midwinter Clinical Course for the combined specialties. The Club was now incorporated and Dr. Jones continued to act as chairman of its Committee, an office he had occupied from the beginning and was destined to hold until 1955. Guest lecturers have been brought from Great Britain, Austria, Switzerland and other Europen countries; from India, from many medical centers in this country, and from Canada.

Dr. Jones enjoyed his membership in the following socities: Diplomat certified by the American Board of Otolaryngology, Los Angeles County Medical Association—President of the Eye and Ear Section in 1937; American Medical Association; American Laryngological, Rhinological and

Otological Society, Inc.—Vice-President (Chairman of Western Section), 1923; American Otological Society; Association of Military Surgeons of the United States; Pacific Coast Oto-Ophthalmological Society; American College of Surgeons; American Academy of Nutrition—member of the Editorial Board of its Journal of Applied Nutrition; Editorial Board of THE LARYNGOSCOPE; Research Study Club of Los Angeles—chief organizer and chairman of its Committee 1928 to 1955.

In Saint Paul's Cathedral there is a plain tablet erected to the memory of the church's great architect, Sir Christopher Wren, with the simple words: "Lector, si monumentum requiris, circumspice." In the hearts of the members of the Research Study Club there may fittingly be erected such a tablet with such an inscription, honoring him who was in a real sense its founder and architect.

* * *

"Seest thou a man diligent in his business? He shall stand before kings." *Proverbs* 22:29. H.S.M.

IN MEMORIAM

ISAAC HAMPSHUR JONES, M.D.*

Dr. Isaac Hampshur Jones, in his prime and even mature years, would want no tears, no sadness, on this occasion; he would remind us that solace has an old meaning: to make cheerful, to entertain, to amuse. Dr. Jones was a master at giving this kind of solace. He loved life, as he loved truth, beauty, and good cheer. He would have us meet death as Socrates did, with courage and a cheerful countenance.

Dr. Jones would have us recall from the Phaedo that after Socrates had drunk the hemlock, his close friends, Crito, Apollodorus and others, were unable to restrain their tears. Socrates alone retained his calmness. "What is this strange outcry," he said. "I sent away the women mainly in order that they might not offend in this way." . . . and he walked about until, as he said, his legs began to fail. He began to grow cold from the feet upward, and when the coldness reached his groin, he said (and these were his last words): "Crito, I owe a cock to Asclepius; will you remember to pay the debt?"

In this same calm and cheerful manner Dr. Jones moved through life and even into the portal of the hereafter. He probably greeted St. Peter with an amusing and appropriate story. His last words to me, a few months ago, spoken at only a decibel above a whisper, were in story-form. He was a superb story teller; he had an apt one for every occasion; even a telephone conversation with him rewarded you with at least one good story.

Dr. Jones has fashioned mansions and monuments that will long survive him. Even his earthly mansions were built of materials—stone and bronze and oak—to stand a thousand years: but he built even more stately mansions. *Flying Vistas*, a book he wrote in the thirties, as a tribute to the work of his colleague, Dr. Theodore Lyster, whom he credited with the creation of a new specialist, the Flight Surgeon, is a monument to Col. Jones, no less than to Gen. Lyster,

*Eulogy delivered by Vern O. Knudsen, Vice Chancellor and Dean of the Graduate Division University of California, Los Angeles.

for devising tests for screening pilots who *are,* from those who *are not,* fit to fly. The subtitle for *Flying Vistas* is *The Human Being, as Seen Through the Eyes of the Flight Surgeon.* The book treats the seeing, equilibrating and motion-sensing functions of the pilot as indispensable parts of the airplane in safe and effective flight. Chapter III, for example, deals with man's eyes and their role in flying. Note how Isaac Jones, the teacher and writer, begins this chapter. I quote:

> All of us old Amherst men sing to this day:
>
> *Here's to 'Tip with dismantled dome—*
> *He never was known to use a comb.*
>
> "'Tip' Tyler taught biology—there was wisdom in that vast bald head. We freshmen soon found out that we had to learn—'Ontogenesis is a brief recapitulation of Phylogenesis.' If you could say that, you passed; if not, you flunked."

More scholarly than *Flying Vistas* are two other books, which are largely the work of Dr. Jones: *The VIII Nerve,* which deals with the physiology and pathology of the internal ear and its associated nervous system, and his earliest book, *Equilibrium and Vertigo.*

Dr. Jones liked to work, and especially to write, with his colleagues. During the ten years that he and I collaborated, he and his co-authors published at least a score of clinical and scientific papers, concerned mostly with impaired hearing—how to diagnose it; and how to correct it. In his later years, his scholarly interests were mainly in vitamins and their applications to otolaryngology. In collaboration with such colleagues as Eugene R. Lewis, Leland Hunnicutt, Jerome A. Hilger and H. S. Muckleston, he prepared annual reviews on vitamins and nutrition. The last one was published in THE LARYNGOSCOPE in December, 1955.

Dr. Jones was a distinguished practicing otologist and organizer as well as a prolific writer for medical journals. He was often consulted by other otologists when they were unsure about their diagnoses or treatments. He organized the Los Angeles Study Club and the Mid-Winter Clinical

Course more than 30 years ago. The latter now attracts more than 300 Eye-Ear-Nose-and-Throat Specialists to Los Angeles each January for a two-week postgraduate course. Many of the world's foremost ophthalmologists and otolaryngologists have lectured and conducted clinics at these annual sessions. Through all the planning and conducting of these sessions the effective guidance of Dr. Jones was evident, but Dr. Jones was modestly in the background.

He and his life-long friend, the late Regent Edward A. Dickson, played a key role in founding the Tennis Patrons' Association, of which Dr. Jones was long the President.

Dr. Jones lived in a home of beauty, culture and refinement, where mutual love between him and his late wife Emilie, and his son Austin, and Austin's wife Fernande and their two children, contributed much to his aboundant life. Besides this good life at home, Dr. Jones enjoyed the respect, devotion and affection of a very large circle of friends, colleagues, and patients. He enriched the treasures of his own mind and heart by living and sharing with others, *but he gave more than he received.* He loved almost everything, from the cooking and stories of Pierre, the Basque chef at the Beach Club, to the finest prose and poetry. I close by reading one of his favorite sonnets, written by a U.C.L.A. colleague, the late Professor of Art, George J. Cox. The title is *Survival*:

When from the poles eternal ice sweeps down
To fold now tropic lands in lunar sleep,
And this dead earth, as a new risen moon,
On other planets other tides shall keep;
When the exhausted sun in cindrous gloom
Threads a dark orbit through unlighted stars,
And the slow grind of timeless years consumes
In silent night all trace of senseless wars;
When all the art and science of mankind
Are with the dinosaur alike decayed,
Brave pioneers of better worlds shall find
Unravished loveliness no greeds betrayed—
For of the triumphs of the human mind
Surely the beauty of truth is saved.

THE RESEARCH STUDY CLUB OF LOS ANGELES

Announces its Twenty-Sixth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 14 through January 25, 1957.

For "Ear-Nose-Throat," the main Guest Lecturers will include Dr. Louis H. Clerf, Dr. George W. Shambaugh, Jr., and Dr. Maurice H. Cottle.

Mrs. Spencer Tracy, Founder and Director of the John Tracy Clinic of Los Angeles for Deaf and Hard-of-Hearing Children and Their Parents, will speak to the "Ear-Nose-Throat" group the opening day of the Convention on "The Role of Parents in the Education of a Deaf Child."

Dr. H. Russell Fisher, a pathologist of note, will bring us up-to-date in his field.

Instruction Courses in Otolaryngology will be provided by three main speakers, Dr. Clerf, Dr. Shambaugh and Dr. Cottle, as well as by Dr. Aram Glorig, Jr., and Douglas E. Wheeler, Ph. D., both of Los Angeles and authorities in Audiometry.

The main Guest Lecturers for "Eye" program will be Dr. John M. McLean and Dr. Graham Clark, both of New York City; and Dr. Samuel J. Kimura of Berkeley, Calif.

Instruction Courses in Ophthalmology will be conducted by Guest Speaker, Dr. Kimura; Dr. Julian N. Dow, Dr. Robert V. Shearer, Dr. Alfred R. Robbins, and a symposium composed of Dr. Clarence H. Albaugh, Dr. John P. Lordon, and Mr. Russell L. Stimson—all of Los Angeles.

The first week will be devoted to the "Ear-Nose-Throat," from Monday to Friday, January 14 through January 18. The "Eye" week will cover Monday through Friday, January 21 through January 25. Those who confine their work to only one of these specialties may complete either subject in one week, or remain for the full two weeks.

As a period of relaxation, Wednesday afternoons will be

free for sight-seeing, shopping, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

Motion pictures of "Ear-Nose-Throat" subjects will be shown during the first week—Monday, Tuesday, Thursday and Friday, 3:00 to 4:00 p.m.

"Eye" subjects will be presented on Wednesday afternoon of the second week for those who prefer to take advantage of this opportunity to view films dealing with subjects in Ophthalmology.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting in San Francisco between the two weeks of the Convention on Saturday afternoon, January 19, and Sunday morning, January 20. This will permit members to attend the Triologic meeting and return to Los Angeles for the "Eye" week. Programs of the San Francisco meeting will be available at the registration desk at the Elks Club.

On Monday evening, January 21, the annual banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of the "Eye" week. This will take place at 7:00 p. m. in the El Venado Room of the Elks Club.

The Women's Entertainment Committee will arrange for various events of interest to the wives of visiting members during the Convention. This Committee will appreciate information as to whether your wife will attend with you, which will give the Committee an estimate as to the number of wives for whom to plan entertainment. Details of all such events will be printed and available at the registration desk.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. R. B. Philbin, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail. The Elks Club is the locale for the entire daily program.

All applicants must be members in good standing, of the American Medical Association, *in order to become eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. These dues are an income-tax-deductible item, as they represent an Annual Membership Fee. Make your check payable to "Mid-Winter Clinical Convention," and mail to Norman Jesberg, M.D., Treasurer, 500 South Lucas Avenue, Los Angeles 17.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D. C., May 5-10, 1957.

The Officers and the Organizing Committee of the Sixth International Congress of Otolaryngology cordially invite you to attend the Congress which will be held in Washington, D. C., U. S. A., May 5 through 10, 1957.

REGISTRATION AND OPENING.

Any qualified physician may become a Member of the Congress by paying a registration fee of $25.00, U. S. A. Non-medical personnel may register as Associate Members for a fee of $10.00, U. S. A. The registration fee includes the privilege of attending all official meetings of the Congress except the banquet for which an additional charge will be made. Those planning to attend the Congress should complete *Form A* and return it to the General Secretary.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill. ....
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, Boston, Mass.
First Vice-President: Dr. Henry M. Goodyear, Cincinnati, Ohio.
Second Vice-President: Dr. Robert E. Priest, Minneapolis, Minn.
Secretary: Dr. Harry P. Schenck, Philadelphia, Pa.
Treasurer: Dr. Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Percy Ireland, Toronto, Canada.
President-Elect: Dr. Lewis F. Morrison.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Washington, D. C., May, 1957.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 13-19, 1957.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland, Ohio.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 6-12, 1957.

THE AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1957.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Irvin J. Fine, 506 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. William Schwartz, 224 Lexington Ave., Passaic, N. J.
Meetings: Quarterly.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracure, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Chevalier L. Jackson.
Vice-President: Dr. John J. O'Keefe.
Treasurer: Dr. Joseph P. Atkins.
Secretary: Dr. Louis E. Silcox.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Harry P. Schenck, Dr.; Benjamin H. Shuster, Dr. William A. Lell, Dr.; William J. Hitschler.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Raymond Kerwin, 310 So. Michigan Ave., Chicago, Ill.
Vice-President: Dr. Frank Wojniak, 6132 So. Kedzie Ave., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. H. K. Rouse, 1300 27th Ave., Gulfport, Miss.
Vice-President: Dr. A. J. McComiskey, 3420 Prytonia St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18, 1957.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Norman Eaddy, Sumter, S. Car.
Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.
Vice-President: Dr. Curtis D. Benton, Jr.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ludwig A. Michael, 3707 Gaston Ave., Dallas, Tex.
Vice-President: Dr. Hal W. Maxwell.
Secretary-Treasurer: Dr. Edward A. Newell, 1511 No. Beckley, Dallas 8, Tex.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Leland R. House, M.D.
Secretary-Treasurer: Sol Rome, M.D.
Chairman of Opthalmology Section: Stephen J. Popovich, M.D.
Secretary of Ophthalmology Section: Richard Kratz, M.D.
Chairman of Otolaryngology Section: Harold Boyd, M.D.
Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P. M. first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting: April 7-11, 1957.

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Col. Wendell A. Weller.
Secretary-Treasurer: Major Stanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.
Time and place of meeting: October 1957, Palmer House, Chicago, Ill.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUDGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. G. M. T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Banff Springs Hotel, Banff, Canada, June 17-19, 1957.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Theodor Hunermann, Dusseldorf, Germany.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congrèss of Bronchoesophagology, Philadelphia, May 12-13, 1957.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salavador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury. Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco de Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filno, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Apartado Postal 21815
Mexico, D. F.

Honorary President: Dr. Ricardo Tapia y Fernández.
President-Elect: Dr. Miguel Arroyo Guijosa.
Secretary: Dr. Luis Vaquero.
Secretary-Elect: Dr. Benjamin Macias Jiménez.
Treasurer: Dr. Rubén Mingram Camargo.

THE NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. J. Kuilman, Surinamestraat 19—Den Haag.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1—Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac Obrechtstraat 66—Amsterdam.
Librarian: Dr. P. G. Gerlings, J. J. Viottastraat 4—Amsterdam.
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